OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

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VIRGINIA MEDICAL MONTHLY

UNIVERSITY OF MINNESOTA RECEIVED JUN 19 '51 LIBRARY

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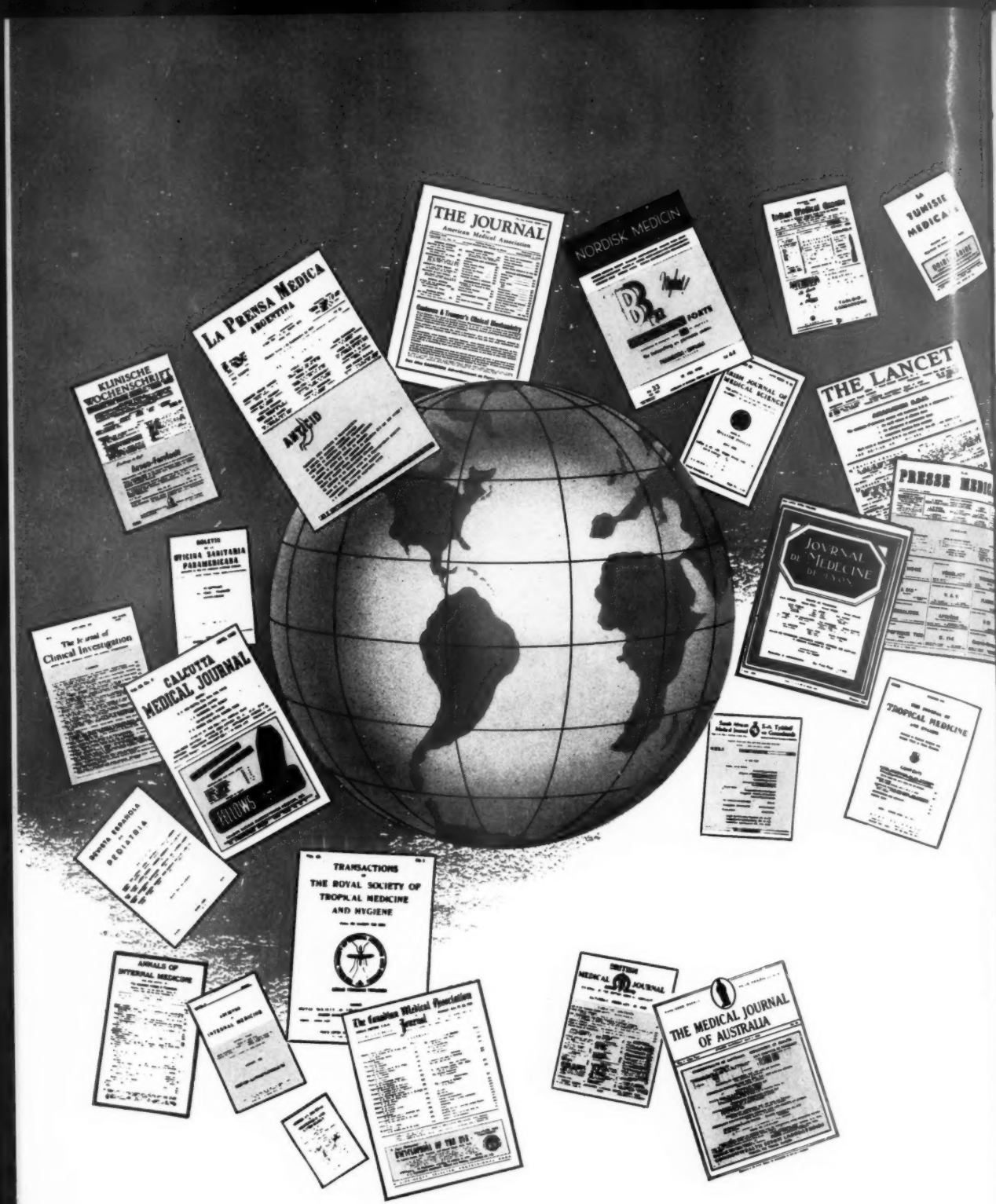

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Vol. 78, No. 6
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RICHMOND, VA., JUNE, 1951

$2.00 A YEAR
25 CENTS A COPY

GUEST EDITORIAL

Advances in Heart Disease in This Half Century

MANY of the laity believe that physicians talk much about heart disease but do little to cure it. A brief review of the advances made in this half century, both in the diagnosis and treatment of heart disease, may serve in part to answer this criticism.

In 1902 Sir James Mackenzie made notable progress in the explanation of cardiac irregularities with simultaneous recording of the jugular, venous, and radial arterial pulses with the polygraph. In 1903 Einthoven of Holland introduced the string galvanometer for electrocardiography which has proved of outstanding value in the diagnosis of myocardial infarction as well as in the clarification of the precise nature of arrhythmias. In 1908 Maude Abbott of Canada published a monumental clinical and pathological study concerning congenital heart disease which laid the ground work for successful treatment of certain forms of this disorder. J. B. Herrick of Chicago published his famous paper, "Clinical Features of Sudden Obstruction of the Coronary Arteries," in 1912, and made possible the modern treatment of myocardial infarction. In 1918 Frey of Germany introduced quinidine as a treatment of auricular fibrillation. In 1920 Pardee of New York City pointed out the correlation of "Coronary" T waves of the electrocardiogram with myocardial infarction. Saxl of Austria in 1920 introduced mercurial diuretics which have proven of tremendous value in the treatment of congestive heart failure. In 1927 Hering of Germany pointed out the significance of the carotid sinus reflexes which has made possible the relatively successful prevention of syncope due to this cause. In 1929 Churchill of Boston successfully performed pericardial resection for chronic constrictive pericarditis. Wilson of Ann Arbor in 1930 introduced chest leads in electrocardiography and clarified the significance of bundle branch block. In 1934 Goldblatt, then in Cleveland, demonstrated the production of hypertension by induced renal ischemia in dogs. In 1934 Beck of Cleveland devised an operation to bring new blood vessels to the heart diseased by coronary arteriosclerosis through pericardial implants of pectoral muscle. In 1935 Smithwick of Boston modified Peet's operation of splanchnic resection for hypertension, a procedure which in carefully selected patients has proved helpful. In 1939 Gross of Boston did the first successful ligation for patent ductus arteriosus. This operation has already saved the lives of hundreds of children. Improvement in the surgical technique for the treatment of mitral stenosis, suggested first by Cutler in 1924, now yields promise for the relief of mitral and aortic stenosis by direct operative treatment of the affected valves. Angiocardiography has been one of the most valuable techniques in the diagnosis of congenital heart disease and vascular disease. The technique first described by Robb and Steinberg in 1940 has been of particular value in the exact diagnosis of congenital heart abnormalities. The anticoagulants, heparin and dicumarol, introduced in 1941 for the treatment of myocardial infarction and pulmonary embolism, have proven of great value in preventing extension of thrombosis and of thromboembolic complications. Subacute bacterial endocarditis, formerly 99% fatal, can now be successfully treated

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in a large proportion of cases by proper chemotherapy. The ballistocardiograph, an instrument which registers graphically the recoil of the heart and the body and the impacts of the ejected blood, was shown by Starr in 1942 to be of value not only in measuring the output of the heart but also in correlating abnormal patterns with heart disease which promises to give important information not obtained by any other form of cardiac examination.

Cardiac catheterization may be accomplished by the introduction of a flexible radiopaque catheter through a peripheral vein into the right auricle, right ventricle, and pulmonary artery. The pressures in these chambers may be measured and samples of blood obtained which are studied for the oxygen content. This procedure, performed by Forssman of Germany upon himself in 1929 and perfected by Cournand of New York, has greatly facilitated the accurate diagnosis of complicated congenital disorders. The brilliant operative treatment of the blue baby with the tetralogy of Fallot was first performed in 1945 by Blalock of Baltimore upon the theoretical recommendation of Taussig, also of Johns Hopkins. In this procedure, anastomosis of a subclavian or innominate artery is made to the right or left pulmonary artery, which enables a large proportion of the blood to be oxygenated by the lungs. At present, the following congenital cardiac lesions are amenable to surgical treatment: (1) coarctation of the aorta, (2) patent ductus arteriosus, (3) tetralogy of Fallot, (4) anomalies of the great vessels producing dysphagia lusoria such as double aortic arch, (5) pulmonic stenosis or tricuspid atresia with patent foramen ovale, (6) pulmonary or systemic arteriovenous aneurysms.

Stead of Duke University has advanced a new theory concerning the fundamental mechanism of congestive heart failure with emphasis on reduction in cardiac output causing reduction in renal blood flow, which in turn causes retention of sodium and increased blood volume, which in turn raises venous pressure. Low sodium diets in congestive heart failure thus have a rational basis. The so-called high output types of heart failure encountered in thyrotoxicosis, anemia, heart failure due to arteriovenous aneurysms, patent ductus arteriosus, and beri beri heart disease can all now be cured by appropriate treatment. Radioactive iodine seems promising in the treatment of certain patients with intractable angina pectoris as suggested by Blumgart of Boston in 1949. In 1950 Pronestyl, a procaine derivative, has been successfully used in the treatment of ventricular tachycardia. In 1950, by the injection of radiopaque dye through a catheter in the aorta, interesting studies concerning the coronary artery circulation have been made.

In the future of cardiology are many unsolved problems, the most pressing of which is the prevention of successful treatment of arteriosclerosis since this disease is the predominant cause of coronary artery disease in middle or later life. Also lacking is precise knowledge regarding the causation and treatment of hypertension. Finally, there is the urgent need for additional information concerning the etiology and treatment of acute rheumatic fever. The temporary control of the rheumatic state with ACTH and Cortisone is a hopeful indication for future progress in the treatment of this disease.

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TOXIC REACTIONS OCCURRING WITH ACTH AND CORTISONE THERAPY

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The preparations ACTH and Cortisone are relatively new, yet they have been used for a large number of conditions and have received considerable publicity in the daily press.

Until recently these preparations have not been available for general use, but in the past few weeks, they have been released and may be obtained on prescription. In view of this, it is important that physicians become fully aware of the severe reactions which may occur during the administration of these drugs.

The pituitary adrenal corticotrophic hormone, ACTH, is prepared from hog pituitary, the concentration of sheep and beef pituitary being very low. The yield, even from hog pituitary, is so small that each patient requires the daily administration of the amount obtained from 100 glands. Cortisone is a physiologically active adrenal cortical steroid originally named "Compound E." It is the principal member of the 11-oxisteroid 17-ketosteroid group. As is not uncommon when powerful new preparations are discovered, the value of these substances is soon over-emphasized in the popular press and in professional literature as well. On the other hand, the toxic effects do not receive attention until much later. During the past year, however, various articles have appeared in the literature describing toxic effects of these two preparations. It is our purpose to call attention to some of these effects with special emphasis on the psychiatric conditions which may develop. But, first, let us discuss briefly the history of these preparations.

In the Fall of 1946 the Armour Laboratories made available a preparation of ACTH (adrenocorticotrophin) to ascertain the properties of this hormone in the human being.

During the succeeding two years, the effect of stimulating the adrenal cortex with ACTH (Armour) was studied in normal human beings and individuals with obvious endocrine defects by a number of investigators.

Practically all of these studies were metabolic balance studies designed to obtain a maximum amount of information concerning adrenal cortical function from the very limited supply of ACTH which was available. As a consequence, a substantial body of information has been accumulated concerning the physiologic and metabolic effect of stimulating adrenal glands in normal human beings and individuals with obvious endocrine defects.

During this first two year period, two disease syndromes in which there may be a metabolic defect were studied, namely, myasthenia gravis and gout, the first by Soffer[1], and the second by Cohn and Wolfson[2]. These investigators found that the clinical status of the patient was materially altered as a result of adrenal cortical stimulation by ACTH (Armour).

During the latter portion of this first two year period a number of groups were exploring the metabolic function of the adrenal cortex in, what were called at the time, "miscellaneous diseases," but there was no thought at the time that stimulation of the adrenal cortex by ACTH (Armour) would alter the clinical course of any of the syndromes under investigation. However, definite improvement was observed by several investigators in diseases under the conditions experiments were being done.

In December, 1948, Dr. Edith B. Farnsworth[3] requested ACTH to study the effect of adrenal cortical stimulation in nephrosis, in which she hypothesized a metabolic defect in adrenal cortical function, and found, in fact, that the clinical status of the nephrotic patient was substantially altered as a result of the hormone administration.

In the latter part of January, 1949, Dr. Philip Hench requested ACTH to study adrenal cortical function in rheumatoid arthritis in line with his

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hypothesis that there may be an adrenal cortical defect underlying this disease. The results observed are, of course, now widely known.

The work of Hench, Kendall, Slocomb and Polley[4], showing that both Compound E (Cortisone) and adrenocorticotrophic hormone (ACTH) dramatically relieved rheumatoid arthritis, was a contribution of great importance, so great in fact that Kendall and Hench have recently been awarded the Nobel prize in medicine for their achievement, each receiving a third interest in the prize, the third recipient being Dr. Reichstein, of Basil, Switzerland.

In October, 1949, Armour and Company invited investigators to report their findings at a two-day meeting. The actions of ACTH and Cortisone in relation to a variety of diseases involving over 800 patients were discussed there. This variety of diseases included 45 different clinical entities; however, we will limit our discussion to the neuropsychiatric disorders in which these preparations have been used.

The adrenocorticotrophic hormone has been used by investigators in specific fields of neuropsychiatric disorders. A group at the Allan Memorial Institute of Psychiatry and McGill University, Montreal, made experiments employing from 2.5 to 25 mgm. doses of ACTH in thirty patients in whom anxiety was the most prominent feature[5]. Isotonic saline was used as a control. It was found that there appeared to be a wide individual variation in response to small doses of ACTH. It was also noted that the control injection of isotonic saline with test situations exactly similar to that under which ACTH was given, produced a response similar to that produced by ACTH, especially in those patients who clinically appeared to have the most severe anxiety. This group, however, was primarily interested in the empirical response, rather than actual treatment of anxiety states.

Pincus and Hoagland[6] at the Worcester Foundation used the administration of from 100-200 mgm. doses in three schizophrenics, expected to do well on shock, coupled with studies made on four schizophrenics receiving placebos. They found no significant improvement in two of the patients receiving ACTH, while one showed marked improvement in two to three weeks. He became less withdrawn and better acclimated to ward routine and began to mingle with other patients. This improvement ceased, however, and he relapsed to his former state one week following cessation of treatment. While this work, of course, is not conclusive, their paper contains some rather significant statements which are quoted as follows: "We have also found that favorable prognosis in electroshock therapy of schizophrenic patients is correlated with the degree of responsitivity of the adrenal cortex to pretreatment doses of 25 mgm. of ACTH." "It is possible that all shock therapies (electric, insulin and metrazol) act as repeated violent stresses to discharge excessive amounts of ACTH which thereby activates the patients' sluggish adrenals inducing consequent beneficial metabolic changes."

At New York University and the Bellevue Medical Center, James J. Smith[7] has used ACTH in the treatment of Korsakoff psychosis, acute alcohol intoxication and delirium tremens. All patients in the series received 25 mgm. of ACTH intramuscularly every six hours and received no other medication or supportive measures either before or during treatment with ACTH. Sedation was also withheld.

There was no apparent clinical improvement in two patients with Korsakoff psychosis. There was some diminution in excitement, but all other symptoms remained essentially unchanged.

Two female patients who were acutely alcoholic showed no change on the ACTH regime. However, there was striking improvement in those patients with delirium tremens. With conventional treatment no improvement was noted before forty-eight to seventy-two hours, while with treatment with ACTH improvement was noted within three to ten hours.

It was also noted that the improved appetite and improved sense of well-being seen as a non-specific response to ACTH might be of value in treatment of patients with anorexia nervosa.

In neurological disorders, ACTH has also been used experimentally. Eight patients with myasthenia gravis[8] were treated with 20 to 25 mgm. doses every six hours for five days. Undesirable symptoms of malaise, occasional abdominal cramps, some respiratory difficulty and some EKG changes occurred, and these disturbances became more marked toward the end of the therapy and continued for one to two days following cessation. After this these symptoms disappeared, some improvement of the symptoms of myasthenia gravis occurred, consisting of increase in appetite, decreased fatigability and weakness,

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and smaller requirement of neostigmine bromide. This improvement was maintained for many months.

Soffer *et al.* administered forty milligrams of ACTH daily in four divided doses for four days to a patient with myasthenia gravis with a thymic tumor who was on prostigmine therapy. During the period of ACTH administration there was no change in the patient's symptoms or in the daily prostigmine requirements; however, three or four days after ACTH was discontinued, an increase in strength was noted, and the daily need for prostigmine became progressively less so that forty-six days after cessation of therapy the patient had required no prostigmine for over a two-week period. A decrease in the size of the thymus was noted following ACTH therapy but with a return to its previous size within two weeks.

It is further reported that in two patients with myotonia atrophica treated with ACTH some subjective improvement in symptoms was noted, but examinations could show no increase in muscular function during hospitalization[9]. After return home, however, it is stated that there was less fatigue. There was no change in mental status. In two patients treated with progressive muscular dystrophy no improvement was found.

Other investigators[10] have also treated two patients with multiple sclerosis, two with Parkinsonism, one with amyotrophic lateral sclerosis, one with progressive muscular atrophy, and one with progressive muscular dystrophy. These patients received totals of from 200 to 575 mgm. of ACTH given in 25 to 75 mgm. doses intramuscularly four times a day from five to seventeen days. No markedly beneficial results were obtained, but it was noted that in multiple sclerosis there was some improvement in gait, tremor and equilibration while on treatment.

Hoefer and Glasser studied the nature of electroencephalographic and neuropsychiatric disturbances which had been noted in fifteen patients treated initially with ACTH at Columbia Presbyterian Medical Center[11], thirteen on the medical service and two at the New York State Psychiatric Institute, the latter two with a diagnosis of schizophrenia. Of these fifteen, two showed no significant changes in the EEG. All others showed definite changes from moderate to severe in degree, which usually appeared three to five days after treatment had been started and consisted of reduction of amplitude, regularity, and continuity of the basic alpha activity. They also had two cases which had been treated with cortisone; both showed slowing of alpha activity in the EEG. The authors concluded that there is a significant relationship between adrenocortical activity and the electrical activity in the brain. Since prior to the treatment eight of the fifteen showed mildly abnormal EEG, they suggested that metabolic disturbances or other changes of minor degree reflected on activity of central nervous system in these cases. Their work was not confined to the EEG findings alone. They studied the pre-morbid personality characteristics and the mental changes occurring during administration of the drug. In the fifteen studied eight showed no significant abnormal pre-morbid personality features, while five showed emotional instability and four showed schizoid features. Ten of the fifteen showed significant mental changes during the time the drug was being administered. Two of these ten were reported to have had severe reactions. These severe reactions consisted of one stupor state and one manic psychotic reaction. The other changes reported consisted of increased tension and irritability, euphoria, and verbal hyperactivity. All this concludes that there are two factors to be considered in connection with these disturbances. First, an organic mental reaction, and, second, a released psychotic reaction with severe exaggeration of premorbid trends. An example of the first is the stupor state of one of the patients and the correlated EEG abnormalities; an example of the second being the psychotic manic reaction.

The manufacturers of cortisone warn that "While definite improvement in mental attitude of patients receiving cortisone usually occurs, in certain instances an exaggerated sense of well-being, and infrequently a manic state may be produced. Mental depression has been reported. In rare instances, preexisting or latent mental derangement seems to have been intensified or precipitated. The mental changes may occasionally appear during the first few days of therapy. Premonitory signs and symptoms of psychotic reactions occur, such as pronounced swings of mood and insomnia."

Likewise, Armour and Company advise against the use of ACTH in "Known psychotics and psychopathic personalities." In recent months we have had the opportunity of observing a psychotic reaction, apparently precipitated by cortisone, and another apparently precipitated by ACTH.

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Brief summaries of these two cases are as follows:

A 51-year old white male was admitted to the Medical College of Virginia Hospital on 7-25-50. History revealed that the patient had had rheumatoid arthritis for three years and that he had been treated since 7-9-50 with cortisone for this condition. He received his first dose of cortisone on 7-10-50 and the last dose on 7-24-50. His son, who was the informant, stated that the patient experienced a feeling of well-being and elation soon after cortisone therapy was started. By 7-18-50 he had episodes of laughing and weeping, and stated to his brother that his whole life was passing before him. Following this, over-activity and over-production of speech progressively increased. However, there were several periods of lucidity lasting for approximately one-half hour. During these times he expressed the idea that his abnormal episodes were resulting from cortisone. On 7-22-50 the patient went to a western movie and during this time began to weep and gave his son all of his money, demanding that he memorize the serial numbers on the bills. He continued to become progressively worse and began calling himself "the cortisone kid." He also was reported to have had some hallucinatory experiences and was referred to the Medical College of Virginia Hospital Emergency Room because of the above stated behavior. Additional history was later obtained, revealing that under physical stress the patient had previously had several transient episodes of vague hallucinations.

On admission the patient was a well developed, white male with a tremor of arms and legs. His speech was spontaneous and irrelevant with much over-productivity. His mood was one of elation. He stated that he was "Adam" and that he had control of the atom bomb. He was completely disoriented and retention and recall could not be tested because of inattentiveness. Insight and judgment were absent. The physical examination was necessarily done with patient under sedation. The examination was essentially negative except for findings consistent with early rheumatoid arthritis. Sedimentation rate on 7-26-50 was elevated. Otherwise, laboratory findings were within normal limits.

The patient continued to be overactive and overtalkative with much delusional content. He was assaultive, combative, destructive, aggressive, and hostile until electric shock therapy was instituted. However, he showed marked improvement after the first two electric shock treatments but this improvement did not hold. Electric shock was continued and after five treatments, he again showed marked improvement, becoming rational, quiet, cooperative, and aware of the circumstances surrounding his hospitalization.

He was transferred to a Veterans Administration Hospital on 8-3-50, where he received further electric shock therapy and was discharged on 9-12-50, as improved.

The second case is a 64-year old white female, admitted to the Medical College of Virginia Hospital on 10-2-50. History revealed that on 9-11-50 the patient began treatment with ACTH for chronic arthritis. She was given ten milligrams four times a day for eleven days while hospitalized in a local hospital in her hometown. The ACTH apparently improved her arthritis, allowing her to enjoy some movement of joints which had previously been painful and ankylosed. However, it was noted by the family that toward the end of her hospitalization, she became somewhat euphoric and also expressed some unusual ideas as manifested by the fear that the "hospital might catch on fire." She also stated that she could hear a patient in the room above her who was "not in his right mind and might come down to my room." A relative to whom she was closely attached died while the patient was hospitalized, and she appeared to express no regrets or sorrow regarding this. She showed a progressive verbosity, became forgetful, and would often lose the trend of her thought. On her discharge from the local hospital, she continued to be over-talkative, forgetful, and circumstantial. She would take long periods to do minor acts, spending as long as twenty minutes brushing her teeth. Approximately one week prior to admission to the Medical College of Virginia Hospital, the patient took as long as two hours to eat a meal. Five days prior to admission she showed echolalia and paralalia and began to suffer some insomnia. For three days previous to admission, she showed some depression and almost constantly repeated the word, "why." The patient exhibited tenacious holding of thoughts and words, continuing to express a word in many variations as follows: "Is anyone going to check that? When it is checked, it will be checked, and then after it is checked, I will double-check it so that it will be checked." She also complained of olfactory hallucinations. She received

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sedation from her local physician and on the morning of admission to the Medical College of Virginia Hospital was unable to recognize her family.

A study of the patient's past psychiatric background revealed that no childhood neuropathic traits were known. The informants stated that the patient had always been a gentle, undemanding person who had depended on others for her decisions and had shown helplessness in the face of the smallest problem. The patient was said to have been timid, but had borne her troubles with great fortitude and had not burdened others with the pain and suffering concomitant with her severe arthritis.

On admission to the Medical College of Virginia Hospital the mental status was as follows: The patient was a small, chronically ill, emaciated individual who was talking constantly and somewhat incoherently. She showed much emotional lability varying rapidly between periods of euphoria and depression. She was able to answer simple questions with simple statements. She exhibited flight of ideas and echolalia, and would readily respond to any verbal stimulus, continuing a thought far beyond its logical end. The patient was markedly disoriented.

Physical examination revealed a blood pressure of 224/110, pulse 120, and temperature 101° rectally. Other findings were consistent with a severe arthritis. Laboratory studies were essentially normal. Electrocardiogram was normal except for an occasional ventricular extrasystole. An electroencephalogram was performed on 10-6-50 which revealed an extremely disorganized tracing. There were high amplitude slow waves with fast activity superimposed, some appearing as spikes. It was felt that a conservative course of treatment should be instituted and the patient's response observed before consideration of any specific therapy. She was, therefore, treated with intravenous fluids, vitamins, and general supportive therapy. The patient was incontinent at time of admission. For the first two days of her hospital stay her condition remained essentially that of admission. On 10-4-50, however, she was quieter and did have a fairly lucid period, lasting approximately two hours, during which time she spoke rationally and with coherence. She continued in this manner, showing progressive improvement in her restlessness and over-productivity. Her confusion continued, but did not appear as marked several days after admission.

Approximately a week after admission, the patient was no longer incontinent and on the thirty-first hospital day appeared exceptionally clear and maintained this state. There were some short, transient episodes of confusion but these were not marked. The patient maintained a good level, was able to mingle well with the other patients, and was friendly and cooperative. She was discharged on 11-14-50 as markedly improved.

In both of the above cases, the toxic symptoms developed during the administration of the drugs. However, internists, who have had considerable experience with these preparations, have noted that withdrawal effects are not uncommon. These consist of anorexia, lassitude, weakness, and mild depression. Since euphoria is commonly noted during the administration, these withdrawal symptoms may often be relative rather than actual.

In addition to the psychotic manifestations of toxicity described above, other toxic symptoms of non-psychiatric nature have been noted following administration of ACTH and Cortisone. These include hirsutism, acne, edema, amenorrhea, muscular weakness, impairment of carbohydrate tolerance, negative nitrogen balance, hypochloremic and hypopotassemic alkalosis, weight changes, and rises in blood pressure.

Thus we find that—as is usually the case with powerful therapeutic agents—ACTH and Cortisone may produce a variety of toxic effects. It is the purpose of this paper to call attention to some of the reactions which may occur. No attempt has been made to show the incidence of toxic effects, and it is fully realized that the two cases presented are of no statistical value. That no far-reaching conclusions may be formulated from a study of these cases is evident. However, it is hoped that the above discussion may help to prevent over-enthusiasm and indiscriminate use of these valuable additions to our pharmaceutical armamentarium.

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Cure Heart Condition by Removing Membrane.

A serious heart condition, caused by tuberculous inflammation of the membrane which surrounds the heart, is being cured by two San Francisco doctors through surgical removal of the diseased membrane.

Pericardiectomy—as the removal of this membrane is called—was performed on four patients suffering from tuberculous pericarditis. The doctors report three of the patients are cured and the fourth shows progressive improvement.

The May 5th *Journal of the American Medical Association* carries this report by Drs. Emile Holman and Forrest Willett of the Stanford University School of Medicine and the San Francisco Veterans Administration Hospital.

The mortality rate from tuberculous pericarditis has been high. Tuberculosis germs attacking the membrane around the heart cause it to become thickened and inflamed, putting pressure on the heart. Patients become increasingly disabled by cardiac compression. In the past, patients have been treated by prolonged bed rest.

New Books

The following are among the recent books received at the Tompkins-McCaw Library of the Medical College of Virginia, and are available to our readers under usual library rules,

Apperly—Patterns of disease on a basis of physiologic pathology.

Association for research in nervous and mental diseases—Life stress and bodily disease. Vol. 29, 1950.

Bourru—L'art de se traiter . . . dans maladies vénériennes. 1770.

Collected reprints of the grantees, National Foundation of Infantile paralysis. 1949.

Cornell conferences on therapy. Vol. 4, 1951.

Directory of medical specialists. Vol. 5, 1951.

Horsfall, ed.—Diagnosis of viral and rickettsial infections.

Kemp—The value of hormones in general practice.

Kidd, ed.—The pathogenesis and pathology of viral diseases.

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A SURVEY OF OBSTETRICS BY GENERAL PRACTITIONERS IN A SMALL GENERAL HOSPITAL*

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The Martha Jefferson Hospital in Charlottesville is a 60 bed private general hospital with 16 obstetrical beds. This paper will present a statistical study of the 3,990 deliveries performed there in the past 16 years. Only those cases which had reached 7 months gestation were included in this study. Virtually all of these deliveries were performed by general practitioners on private patients who had been given prenatal care by the physicians doing the deliveries.

Table I gives the general statistics on the entire group. Primiparas constituted almost half of the

TABLE I

Total Number of Deliveries		3990
Primiparas		49%
Multiparas		51%
Age Distribution		
Under 26 yrs.		53%
26-35 yrs.		39%
36 and over		8%
Presentation		
Occiput anterior		93.8%
Persistent occiput posterior		2.6%
Breech		3.0%
Other presentations		.6%
Mode of Delivery		
Spontaneous		77.0%
Forceps		19.9%
Version and extraction		2.0%
Cesarean section		1.1%
Maternal Mortality		.048%
Fetal Mortality		2.7%
Toxemia of Pregnancy		1.1%
Placenta Praevia		.45%
Postpartum Hemorrhage		7.0%
Maternal Morbidity		6.2%
Multiple Births		0.9%
Twins	34	
Triplets	1	

cases and slightly more than half were 25 years of age and under. 93.8 per cent had occiput anterior presentation at the time of delivery, 2.6 per cent were persistent occiput posterior, 3 per cent had breech presentation and .6 per cent had other abnormal presentations. 77 per cent of the deliveries were spontaneous, almost 20 per cent were by forceps, 2 per cent were by version and 1.1 per cent by cesarean section.

The reported incidence of cesarean section varies widely over the country. For the city of Detroit the incidence in 1945 was 2.5 per cent[1], while for the same year Chicago had 3.67 per cent[2]. A recent series from Duke Hospital reports an incidence of 3.8 per cent on private patients and 0.66 per cent on ward cases[3].

There were 2 maternal deaths in our series, constituting a maternal mortality of .048 per cent. One of the maternal deaths occurred during the early part of the period and was caused by hemorrhage and shock due to placenta previa. The other death occurred in 1948. It was due to pulmonary embolism on the ninth postpartum day in a primipara who had been delivered by low forceps.

Our maternal mortality rate of .48 per thousand is better than most reported series. From Bethesda Hospital in Cincinnati a report of 15,088 deliveries gave a maternal mortality rate of 1.7 per thousand[4]. The figure for the state of Virginia in 1947 was 1.7 per thousand and for the entire United States in 1945 was 2.1 per thousand.

The gross fetal mortality was 2.7 per cent. This figure also compares favorably with other series. The Cincinnati series[4] had an infant mortality of 4.5 per cent.

Toxemia of pregnancy was present in 1.1 per cent of the cases and placenta previa in .45 per cent.

Hemorrhage was considered present when the estimated blood loss was 500 cc. or more. This occurred in 7 per cent of our series. The incidence of hemorrhage for the first 10 years of this study was 10 per cent and for the last 6 years was 4.2 per cent. While this is a definite improvement, the figure is still too high. Dieckman at the Chicago Lying-In reports an incidence of 2.5 per cent[5].

6.2 per cent of the mothers had puerperal morbidity, i.e., a temperature of 100.2 degrees by mouth on any 2 postpartum days. This morbidity rate has

*Read before the annual meeting of the Medical Society of Virginia at Roanoke, October 8-11, 1950.

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not improved during the years of this study. In a series of 3,457 deliveries at the Evanston Hospital, Cummings reports a maternal morbidity rate of only 3.64 per cent[6].

There were 34 sets of twins and one set of triplets in the series.

Table II shows the breakdown of the fetal mortality cases. Of the 107 deaths, 66 were stillborn

TABLE II
FETAL MORTALITY

107 cases			2.7%
Stillbirths	66		
Neo-natal deaths	41		
Congenital anomalies	12		
Corrected fetal mortality			2.3%
1934-44	3.5%	1944-50	2.1%

Presentation

	% incident in fetal mortality cases	% incident in entire series
O A	76.0	93.8
O P	7.0	2.6
Breech	15.0	3.0
Others	2.0	0.6

Mode of Delivery

	% incident in fetal mortality cases	% incident in entire series
Spontaneous	69.0	77.0
Forceps	19.0	19.9
Version	8.0	2.0
Cesarean	4.0	1.1

Causes of Infant Death

Hemorrhage or trauma	20	Cord Complications	6
Prematurity	15	Placental complications	5
Anomalies	13	Miscellaneous	35
Unknown	13		

and 41 died within the first 2 weeks of life giving a gross mortality rate of 2.7 per cent. There were 12 cases of congenital anomalies incompatible with life, making the corrected fetal mortality 2.3 per cent. A gratifying decrease in fetal mortality was seen when the rate for the first 10 years of the period covered by this study was compared with that of the last 6 years, the percentage having come down from 3.5 to 2.1. A study of the percentage incidence of the various presentations in the fetal mortality group showed a significantly higher incidence of all of the abnormal presentations. Also the operative deliveries other than forceps occurred about four times as frequently as they did in the series as a whole. The causes of infant deaths were so varied that classification was difficult. Intracranial hemorrhage or obstetrical trauma was the apparent cause in 20 cases, prematurity was the cause in 15, while congenital anomalies and stillbirths of undetermined cause constituted 13 cases each. Complications of the umbilical cord and placenta caused 11 deaths and the remaining 35 deaths were due to a wide variety of causes.

Table III gives percentage rates for infant mortality, hemorrhage and maternal morbidity in the

TABLE III
RESULTS OF OPERATIVE DELIVERIES

	Fetal Mortality (2.7%)	Hemorrhage (7.0%)	Morbidity (6.2%)
Low Forceps	0.8%	8.9%	8.8%
Mid and High Forceps	16.5%	8.7%	33.0%
Version and Extraction	11.4%	25.0%	14.0%
Cesarean	8.7%		8.0%

Figures in parenthesis indicate series incidence of that item.

operative deliveries. The percentage figures in parenthesis are the rates for the entire series. Blood loss in cesarean sections was not determined. Low forcep deliveries had a lower infant mortality and the incidence of hemorrhage and maternal morbidity was not significantly higher. In mid and high forceps the infant mortality and maternal morbidity were greatly increased. In version and extraction all three figures were considerably increased. Cesarean section had a significantly higher infant mortality.

44 cases of toxemia (Table IV) constituted the extremely low incidence of 1.1 per cent. Two fac-

TABLE IV
TOXEMIA

44 cases		1.1%
Eclampsia	9	
Pre-eclampsia	35	
Cesarean Deliveries		
1934-1944		16%
1944-1950		4%
Fetal Mortality		
1934-1944		16%
1944-1950		16%

tors contribute to this fact. The proximity of the University of Virginia Hospital, with its greater facilities and personnel for handling the more complicated cases, results in most of the patients who have had inadequate prenatal care going to this institution. Secondly, all of the patients in this series were private patients and the low incidence of toxemia indicates a high degree of good prenatal care.

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9 cases of eclampsia occurred in this series which is about the same incidence as reported from the Chicago Maternity Center in 1933 where 10 cases were found in 5,486 deliveries[7]. An interesting change in the trend away from cesarean section as a method of delivery is shown in this table. During the years of 1934 to 1944 16 per cent of the toxemia cases were delivered by section, while from 1944 to 1950 only 4 per cent were so delivered.

Fetal mortality in these cases was 16 per cent throughout the series. Apparently little progress has been made in reducing fetal mortality in cases with toxemia.

Persistent occiput posterior (Table V) was present in 102 cases. 44 per cent of these delivered spontaneously without rotating to the anterior position. 17 per cent were delivered with forceps after rotation, while forceps extraction with the occiput still posterior was done in 33 per cent. Version and extraction was done in 6 per cent. Fetal mortality in this group was triple that of the entire series, maternal morbidity was double, while the incidence of hemorrhage was unaffected.

TABLE V

PERSISTENT OCCIPUT POSTERIOR

102 cases	2.6%
Mode of Delivery	
Spontaneous	44.0%
Forceps with rotation	17.0%
Forceps without rotation	33.0%
Version and extraction	6.0%
Fetal Mortality (2.7%)	6.3%
Hemorrhage (7.0%)	5.1%
Maternal Morbidity (6.2%)	12.7%

Figures in parenthesis indicate series incidence of this item.

In Table VI a marked change in the methods used to induce labor is shown. During the years 1934 to 1944, bag or bougie induction was carried out 22 times and rupture of membranes was done 39 times. During the past 6 years bag or bougie was used only twice whereas rupture of membranes was done 204 times. These 204 cases were studied to determine the safety of this procedure as carried out in our hospital. It can be seen that no significant difference is present in fetal mortality, maternal morbidity or the incidence of hemorrhage.

TABLE VI

SURGICAL INDUCTION OF LABOR

Method	1934-1944	1944-1950
Bag or Bougie	22	2
Rupture of Membranes	39	204
Results in 204 Cases of Rupture of Membranes		
Maternal Mortality (.048%)		0
Fetal Mortality (2.7%)		2.5%
Maternal Morbidity (6.2%)		8.4%
Hemorrhage (7.0%)		4.9%

Figures in parenthesis indicate series incidence of this item.

TABLE VII

ANTEPARTUM VAGINAL EXAMINATIONS

Frequency	
1934-1944	19%
1944-1950	7%
Maternal Morbidity (6.2%)	10.5%

Figure in parenthesis indicate series incidence of this item.

The last slide shows a study of the patients who had vaginal examinations during labor. The frequency of this procedure diminished from 19 to 7 per cent during this study, and the maternal morbidity was apparently increased in these cases.

In summary, a statistical study of nearly 4,000 deliveries by general practitioners at the Martha Jefferson Hospital in Charlottesville reveals a low incidence of serious complications and exceptionally low mortality rates.

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DISCUSSION

DR. JOHN O. BOYD, JR., Roanoke: I think all of us have heard many times that statistics can be made to prove anything you want to prove but I don't believe that any of us in this instance could take exception to the manner in which Dr. Daniel has analyzed the cases in Martha Jefferson Hospital. In comparing them with similar statistical analyses of other institutions I have been very much impressed, and I am sure you have, by the favorable way his figures compare. Really it leaves very little for me to say.

There is no discussion I can see except to commend Dr. Daniel and the staff of the Martha Jefferson Hospital for the very excellent way in which they handled their patients as shown by this objective analysis.

I think perhaps there are some lessons we may learn with

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benefit to all of us. To me this analysis is of particular value for the reason that our chairman pointed out, the type of hospital in which this study was made. So many times, and to the best of my knowledge in practically every instance, such reports have concerned themselves with teaching institutions. I think we hold nothing but commendation for the teaching institutions and all they have done for us but I don't believe many of us out in practice can help but appreciate the fact that we have had to learn to get along without a lot they have. We have had to improvise and change things to suit circumstances and we have wondered frequently, at least I have, just what effect this might have on a critical analysis, an objective analysis, of results obtained under other circumstances. I think the opportunity afforded here gives us a partial answer to this and I hope will encourage those of us who are in similar institutions not actively affiliated with the teaching schools to carry out similar studies for our own edification.

Something else that was very important which Dr. Daniel pointed out to us was the availability of expert consultation through the University of Virginia Hospital. He implied a spirit of cooperation between the obstetrical staff there and attendants of the Martha Jefferson Hospital, which is certainly to be desired whenever such consultants are available.

Something further I believe we could gather from this study, at least in explanation of the very favorable comparison between the figures at Martha Jefferson and the other analyses referred to, might be the conservatism that seems to be apparent there. Dr. Groseclose pointed out that one of the modern trends is that men experienced in obstetrics are increasingly recognizing the value of conservatism. In spite of everything we are becoming less radical and conservatism is the thing that counts. The low incidence of Caesarean delivery might well be a factor in the very commendable results that have been obtained at Martha Jefferson Hospital.

I think that the value to the staff of this analysis is tremendous and that they will benefit from it as much as any other individuals concerned. We are indebted to Dr. Daniel and to the staff for making available to us this study.

DR. E. S. GROSECLOSE, Lynchburg: I hope the audience will pardon me for rising again but I don't want to fail to take this opportunity to commend Dr. Daniel for this report. To me it is little less than remarkable. I think his figures are outstanding and I hope that the figures he has given have shown that the trend is now as I tried to present in my paper. To me this presentation is an outstanding report. I, for one, have always been a great admirer of the general practitioner and after seeing and hearing his report, being a specialist, I again want to commend them for the work they have done. I think it is a remarkable series of figures. I think they should be commended by every physician in the State.

QUESTION FROM THE FLOOR: I hope that Dr. Daniel in closing will indicate what rules or regulations, if any, exist in his hospital for consultation in complicated cases,—that is, whether there is consultation between other members of his staff or whether consultants are called from the outside, or whether major operations are done with or without consultation.

DR. DANIEL: Thank you for the discussion and favorable comments.

As to the question on consultations—we have no set policy, no set rules by which we operate the consultation field at our hospital. In the matter of caesareans, there are three general practitioners who do their own caesareans. The rest of us call in surgical help for that. I don't think there is any rule about the performance of a consultation in regard to caesareans, but certainly 95 per cent at least of the caesareans performed were done on patients on whom consultations had been had. Due to the proximity of University Hospital, our consultations have nearly always been with members of that staff rather than with members of our staff.

I am glad Dr. Boyd pointed out, or mentioned, at least, the value of such a study. I would like to make a few remarks about the helpfulness of such a study to any hospital. This presentation here, of course, was only a very small part of the whole study. Actually our own study is divided, even down to the staff members who were involved, so we had precentage figures for each doctor as well as for the entire group, and, while we did not advertise it, we were able to present it so each doctor knew his own figures without being able to recognize the others. It was a great help in that line. This is a type of study which can be carried out in general hospitals with relative ease provided it is properly planned and the mechanics worked out well in advance, and I would like to urge that other small hospitals do it, not necessarily for the value it has for the profession as a whole but for the hospital itself. By using a trained medical librarian, who gives only a small part of her time to this work, each chart on the hospital patient is processed with this study in mind by a punch card system which is very easily worked out. It is important that the plan for the study be well worked out and completely agreed upon before it is started, but it is a method of study and method of analysis extremely important and well worth the effort it takes.

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HYPERSPLENISM IN INFANCY AND EARLY CHILDHOOD: A REPORT OF THREE CASES*

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The concept of hypersplenism as it now stands may be used to explain the pathological physiology seen in a number of different clinical conditions.[1,2] There have been numerous reported cases occurring either as primary or secondary states, but few of these have been in the pediatrics age group. During the past year we have studied three cases of hypersplenism. It is the purpose of this paper to report two unusual cases of panhematopenia and one case of congenital hemolytic anemia in crisis during the fourth week of life.

Hypersplenic syndromes, as described by Doan,[1] Wiseman,[5] and Dameshek,[2] may be manifested as congenital hemolytic anemia, primary thrombocytopenia, splenic neutropenia, or, as occurs in combination, splenic panhematopenia. It is recognized by most investigators that these states may be due to either primary alteration of the splenic physiology, or may be secondary to a number of well-recognized pathological states, either localized in the spleen or generalized throughout the body. The exact mechanism by which the above conditions are produced by the spleen is still not agreed upon by all investigators. Doan, Wiseman, Wright, and others,[3,4,5] believe that they may be due to splenic parenchymal cellular hypersequestration plus an excessive concentration of some lytic enzyme, i.e., lysolecithin, plus reticuloendothelial cell hyperplasia and hyperphagocytosis, in addition to an abnormal cell in some cases. Dameshek,[2] on the other hand, feels that the spleen elaborates an inhibiting substance which acts on the specific cellular elements in the bone marrow. This inhibition of the marrow is reflected in the peripheral blood by a diminution in number of the affected element, whether it be the red cell, granulocyte or platelet. Both of these schools of thought have much experimental and clinical data to support them in their views. In different cases, one or more of these mechanisms may be at work to produce the syndromes known as hypersplenism. Whatever the mechanism may be, everyone is agreed that splenectomy is usually curative in the primary cases and may produce a favorable response in the secondary ones unless one of the contraindications for splenectomy is present, as will be mentioned later.

In general, the diagnosis of hypersplenism rests upon the history—which is dependent upon whether the cellular element involved is the red cell (anemia), granulocytopenia (recurrent infection), thrombocytopenia (bleeding tendencies), or a combination of these;[7] the physical findings—either with or without splenomegaly to support the history; and, probably the most important, the laboratory findings of depression of any or all of these elements in the peripheral blood. The bone marrow may be normal or hyperplastic[6] for one or all of the cellular elements—with or without maturation arrest. Other laboratory data such as serum bilirubin, fecal urobilinogen, reticulocyte counts, fragility tests, may help in evaluating any particular case.

Secondary hypersplenic states may involve processes such as leukemias and similar malignancies, tuberculosis and similar infections, Gaucher's disease, and especially myeloid metaplasia of the spleen, and their place should be thoroughly evaluated.

Splenectomy should never be resorted to where there is diffuse bone marrow damage or aplasia which has caused the spleen to take over a large part of active blood formation, even though at the same time it may be also over-actively destroying blood elements.

CASE REPORTS

Case I: H. C., a five year old colored female was first admitted to the hospital on December 6, 1945, complaining of sore throat, pain in abdomen, vomiting, and pallor of four days duration. Physical examination at that time showed a pale, acutely ill child with a temperature of 101°. There was a greenish-yellow color of the sclerae. A loud systolic mur-

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Read before Virginia Pediatric Society, May 5, 1950, Roanoke, Virginia.

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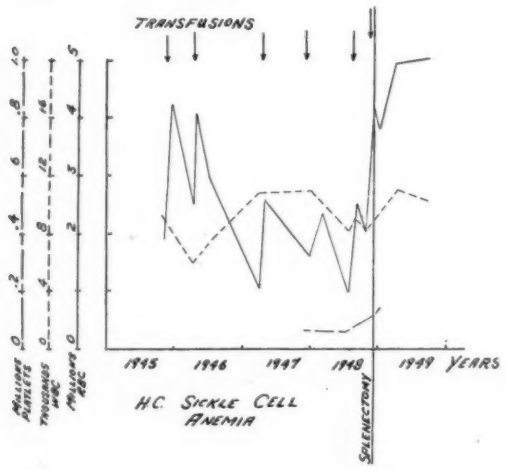
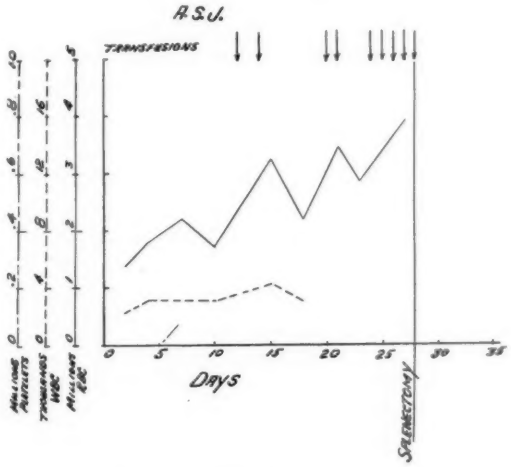
mur was heard over the entire precordium. Apical rate was 140. The spleen was markedly enlarged, extending almost to the midline medially and down four finger breadths below the right costal margin.

Red count at that time was 1.9 million, with a hemoglobin reported as less than 7. Sickle cell test showed 50 per cent sickling at the end of two hours.

Following this admission the child was followed in the outpatient department, somewhat erratically, and was readmitted to the hospital on three occasions between January, 1946, and July, 1948. There





was very pronounced anemia. During these admissions relatively large quantities of whole blood were necessary to produce much change in her blood counts. (Laboratory data included in Chart I.)

On the fourth admission, July 4, 1948, the child was admitted with a history of fatigue, listlessness, and headache, associated with high fever and persistent nose bleed of 24 hours duration. Physical examination on this admission was essentially that of a well-developed, undernourished seven year old child with marked pallor and mild icterus of the skin and mucous membranes. Pulse was 150, temperature 102°, and blood pressure was 110/70. There were several small hemorrhagic areas over the tonsils. The liver was felt two finger breadths below the right costal margin. The spleen extended down to the umbilicus and one to two finger breadths to the left of the iliac crest.

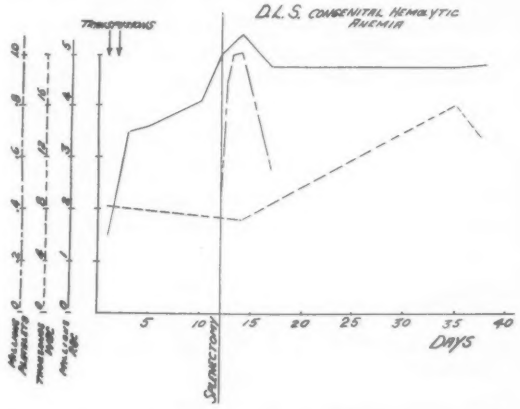
Admission blood counts showed 1 million r.b.c., with 2.5 gm. hemoglobin. Uncorrected white count was 15,000, with a normal differential. The red cells were markedly hypochromic, with marked sickling. During the next six hospital days the patient received multiple transfusions. The first two transfusions were followed by marked febrile and hemolytic reactions. Following these transfusions, the hemoglobin was 3.5 gm. with 1.4 million r.b.c. The child was seen in consultation by the department of hematology, and she was placed on an extract of whole hog spleen. Examination of the bone marrow showed a normoblastic hyperplasia, with an erythroid myeloid ratio of 3. Bleeding time, clotting time, prothrombin time, and fragility test were all normal. Sickling test showed 4 plus immediate sickling. During this time the child was on the spleen extract, but no improvement in the blood picture was noted in the repeated blood counts. The child's hemoglobin varied from 3 to 6 gm., while the r.b.c. ranged between 1.52 and 2.5 million, with up to 42 per cent nucleated red cells. Uncorrected white count remained around 8,000 to 14,000. Platelets on several occasions varied between 42,000 and 60,000, and reticulocyte count ranged from 2.5 to 10 per cent. Fecal urobilinogen values were between 1,040 and 1,452 mg. per 100 gm. feces. The child was discharged on the 31st hospital day afebrile, with 5.8 gm. hemoglobin and 2.5 million r.b.c.

Two months after discharge this child was readmitted, showing no improvement in her blood picture, though she had continued taking the spleen extract. She had been doing relatively well until two days prior to admission, at which time she began running a fever, complaining of abdominal pain, and

for twenty-four hours prior to admission she had had almost a continual nose bleed.

Physical examination showed a poorly nourished, acutely ill, very pale, somewhat icteric colored female, with epistaxis. Temperature was 104.5°, with remaining physical examinations as on previous admissions.

Admission laboratory work showed a hemoglobin of 3 gm., with 900,000 r.b.c. W.b.c. revealed 7,300 with the differential showing 210 nucleated r.b.c. per



100 w.b.c., with a white cell differential normal. During the next two weeks the child was given multiple transfusions, which totaled 14 pints of whole blood. Her hemoglobin never rose above 5 gm. On November 5, 1948, in view of her failure to respond to multiple transfusions, a splenectomy was done, even though most sickle cell anemias were known not to respond to this. However, the size of the child's spleen and the fact that she was showing active blood destruction plus the probability that the huge spleen was trapping large quantities of blood indicated that she would probably benefit. Preoperatively she received 1,500 cc. of blood within a period of thirty-six hours, and during the operation she was given 1,000 cc. The child withstood the procedure well, and her post-operative course was essentially uneventful. Forty-eight hours after operation her hemoglobin was 11.5 gm., with 4.13 million r.b.c. Corrected white count was 3,900. Differential showed 288 nucleated red cells per 100 white cells, with an otherwise normal differential. Following this the child's counts became stabilized at 7 to 7.5 gm. of hemoglobin, with 3.1 to 3.9 million r.b.c. Platelet count was 135,000 per cubic mm. Fecal urobilinogen values a week postoperatively were 800 mg. per 100 gm. of feces. The child was discharged from the hospital asymptomatic on the 41st hospital day.

The child was readmitted to the hospital in March, 1949, for tonsillectomy and adenoidectomy, and she had been asymptomatic since the last discharge. Her hemoglobin was 9.4 gm., with 4.96 million r.b.c. and 11,800 w.b.c., with 1.2 per cent reticulocyte count. Bone marrow done at that time showed a normoblastic hyperplasia, with an M:E ratio of 1.2:1. Bleeding time and clotting time were normal, and clot retraction was good in one-half hour. Indirect van den Bergh showed 0.75 per cent.

The child was seen again in September, 1949, and was found to have been asymptomatic during the interim. Physical examination was essentially negative at this time. Hemoglobin was 9.4 gm., with 5.7 million r.b.c. and 10,800 w.b.c. Reticulocyte count was 2.3 per cent, while the hematocrit was 31.5. Serum bilirubin was 0.66 mg.

Pathology Report: The spleen was enormously enlarged, weighing 1,850 gm. and measuring 25 x 15 x 8 cm. The capsule was smooth except for a large depressed scar on the anterior surface. On section the tissue was brown and firm. Microscopic examination showed a greatly altered structure. The splenic pulp was packed with red cells, many of which showed sickling. The Malpighian bodies were small and consisted of a few scattered lymphocytes. The splenic arteries were very thin-walled. A few scattered cells resembling megakaryocytes were found in the splenic pulp. There was an increase in the splenic connective tissue, which in some areas contained hemosiderin.

Comment: Splenectomy in sickle cell anemia is usually of little benefit,[8] but in this child who was presented at the hospital on several occasions in acute hemoclastic states and who developed thrombocytopenia and neutropenia while being followed and who showed little or no response to transfusions as progression occurred, splenectomy has been of marked benefit. A ten months follow-up has shown stabilization of her blood counts at a satisfactory level and no further episodes of epistaxis. Although hematological data at fifteen months after operation is not available, clinically the child is active, having been converted from chronic invalidism to normal activity by splenectomy.

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Case II: A. S. J., a four year old colored female, was admitted to the hospital with the complaint of a mass in the abdomen of several months' duration. The mother was of borderline intelligence, and had noted only ease of bruising of several months' duration and an upper respiratory infection of two weeks' duration. She was taken to a physician because of the respiratory symptoms, and he referred her to the hospital. Past history and family history were noncontributory to her present illness.

Physical examination at the time of admission showed a thin, four year old female, with marked pallor and extremely small extremities. Weight was 32 pounds, temperature was 101.6° rectally. Except for a loud blowing systolic murmur over the entire precordium and a few shotty axillary lymph nodes, the physical findings of significance were limited to the abdomen. There was a marked visible asymmetry of the abdomen, with bulging of the left side. A firm mass was felt which occupied the whole abdominal left upper quadrant and extended below the left iliac crest and to the midline. The edge was sharp. The liver edge, which was firm, smooth, and sharp, was palpable 1 cm. below the right costal margin.

Laboratory findings on admission showed normal routine urinalysis. Her hemoglobin was 3 gm., with 1.8 million r.b.c., and the total white count was 3,000, with 2 per cent myelocytes, 3 per cent juveniles, 2 per cent bands, 28 per cent PMNs, 60 per cent lymphocytes, and 5 per cent transitional monocytes. Platelets numbered 53,000 and there were 3.2 per cent reticulocytes. There was marked anisocytosis, poikilocytosis, and microcytosis. A concentrate of the peripheral blood showed a few large pathological lymphocytes, but no immature cells. There was granulocytic and erythrocytic hyperplasia of the bone marrow, with an M:E ratio of 5:1. There were also many lymphoid and reticular elements, but very few "blasts" were present. Bleeding time was 14½ minutes with clotting time, by the Lee and White method, of 3½ minutes. There was little or no clot retraction at the end of twenty-four hours. Prothrombin time was 16 seconds, with a control of 13 seconds. A prothrombin consumption test was normal. Icterus index was 33, with a slightly positive direct van den Bergh, while the serum bilirubin was 4 mg. per cent. Total protein was normal. The tuberculin skin test was positive.

Numerous other tests were carried out in an attempt to rule out or confirm other disease processes which produce a secondary hypersplenism. These included histoplasmin skin tests, x-rays of the long bones, chest, abdomen, intravenous pyelograms, bone marrow culture for histoplasma, fragility tests, Coomb's test, and tests for sickling. All of these were normal except for the x-rays of the abdomen and intravenous pyelograms which revealed only the large abdominal mass.

During the several days following admission, the child's blood counts and other laboratory findings remained on repeat studies essentially the same. A diagnosis of hypersplenism with panhematopenia was made, although it was felt that this still might be secondary to some other disease process not otherwise manifest at that time. On the twelfth and fourteenth hospital days the patient was given two transfusions of 250 cc. of whole blood, following which her hemoglobin rose to 6 gm., and the red count was 2.21 million. At this time the child began to show daily temperature spikes to 103.5° rectally which did not respond to penicillin and examination did not reveal any new findings. At the same time her counts began to fall again sharply.

It was decided that splenectomy offered this patient her only hope, and she was given daily transfusions of 250 cc. for four days. Prior to her last transfusion her counts had risen to 9.5 gm. hemoglobin and 3.93 million r.b.c. Bleeding time remained markedly prolonged, and the prothrombin time continued to be elevated (18 seconds with control of 13 seconds) in spite of daily Vitamin K. Although her condition remained poor, it was felt that further delay would gain nothing.

On the 28th hospital day, a splenectomy was done, and at the time of operation bleeding was minimal. However, approximately three hours post-operatively the patient suddenly went into shock. She was placed in oxygen and given 400 cc. whole blood. Her blood pressure rose to 70/50, but while still receiving blood she expired quite suddenly, six hours postoperatively.

Autopsy Report: The autopsy was performed twenty-seven hours after death. Pertinent findings were 940 cc. of unclotted blood in the abdominal cavity. No source for the bleeding was found. All the ligatures on the splenic vessels were secure. There was no evidence of obstruction of the splenic vessels. A small subpleural tuberculous lesion was found in

the apex of the right lung. The associated hilar lymph node was caseous. No other evidence of tuberculosis or other systemic disease was evident. There was a moderate generalized enlargement of all the lymph nodes in the body. The liver showed areas of congestion but was otherwise normal.

The spleen weighed 720 gm. (normal 39 gm.) and measured 18 x 12 x 6 cm. The organ was of normal configuration. The capsule was smooth with no adhesions. The cut surface revealed dark red firm tissue, and the Malpighian bodies were not prominent. On microscopic examination the sinusoids and pulp were engorged with normal appearing red cells. There was moderate increase in the connective tissue. The germinal centers of the Malpighian bodies were active. A few scattered areas of erythropoesis were seen. Very little pigment was seen.

Comment: This child had evidence of blood destruction as indicated by indirect serum bilirubin of 4 mg. per cent which was maintained. All tests to demonstrate a hemolysin in the blood were negative. There was marked neutropenia and thrombocytopenia. Coupled with these three processes was a hyperplastic bone marrow. All efforts to demonstrate a systemic disease that might give these findings were unsuccessful.

The indications for splenectomy seemed quite clear. It is unfortunate that postoperative hemorrhage terminated the case quite suddenly so that proof that her hematological course would have been changed is lacking.

On post-mortem examination no systemic disease or evidence of portal obstruction was found to explain the splenomegaly. Thus it would seem that this patient would be classified as a primary hypersplenism with panhematopenia, and she should have responded well to splenectomy if hemorrhage had not supervened.

Case III: D. S., a five weeks old female infant was brought to the hospital with the complaint by the mother of having done well following a full term normal pregnancy until the age of four weeks, when the diapers were noted to be stained a red color after voiding. This had continued until just prior to the time of admission to the hospital. The urine itself was never noted by the parents, but they felt that the stains were coming from that source. Two days prior to admission the baby began to take its feedings poorly, became very pale, and on the day prior to admission a yellowish tinge to the skin had been noted. The family history was significant in that the father had undergone a splenectomy while in the service because of recurrent jaundice which had required transfusions, often with only transitory benefit. Following splenectomy he had been asymptomatic. It was the father's impression that he had malaria, but examination of his blood smear revealed definite spherocytosis. The rest of the history as obtained was negative.

Physical examination revealed a well developed, poorly nourished seven pound six ounce infant who was extremely sluggish and appeared acutely ill. Her hydration was poor and the temperature was 100.8° rectally. There was marked pallor of the skin and mucous membranes with mild icteric tinge. The apical heart rate was 160 with a soft systolic murmur over the entire precordium. The spleen was felt two centimeters below the left costal margin.

Laboratory studies showed the urine to be completely negative. Hemoglobin was 4.75 gm., with 1.55 million red cells and 8,100 white cells, with a normal white cell differential. Hematocrit was 12 per cent, with cell indices being M.C.V. 77.4 cubic microns, M.C.H.C. 39.1 per cent, M.C.H. 30.3 micromicrograms with a reticulocytosis of 12 per cent. The platelets appeared increased in number. A fragility test showed beginning hemolysis at 0.46 per cent with complete hemolysis at 0.40 per cent saline with a control not beginning until 0.44 per cent and completing at 0.32 per cent. An indirect and direct Coomb's test were negative. The icterus index was 10, with a serum bilirubin of 0.4 mg. per cent.

A diagnosis of congenital hemolytic anemia in crisis was made. The infant was immediately hydrated with glucose and saline solutions intravenously and was given a transfusion of 100 cc. whole blood on admission and on the following day. The hemoglobin rose to 11 gm. with 3.5 million red cells and hematocrit of 35 per cent. The spleen became smaller in size. When the infant maintained its counts at this level splenectomy was deferred until it was felt that conditions were optimal.

On the 12th hospital day a splenectomy was done. The infant received 125 cc. whole blood during the procedure which was tolerated well. On the first postoperative day the counts had risen to 14 gm. hemoglobin, with 5.4 million red cells. The platelet

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count immediately after operation was 413,000 and rose to 900,000 within eight hours and was up to 1,000,000 the next day. No ill effects were noted from this, and on the second postoperative day the platelets began falling and by the fourth postoperative day had returned to 500,000. Following splenectomy the patient's blood showed increased resistance in hypotonic saline, as compared to a normal control on two occasions.

The infant was discharged on the 19th hospital day in excellent condition, weighing nine pounds, three ounces. Two months later she weighed twelve pounds, seven ounces. Blood counts at that time showed 11.5 gm. hemoglobin, with 4.85 million red cells and 13,800 white cells. The differential count was normal, and there were 0.8 per cent reticulocytes.

Pathology Report: The spleen weighed 30 gm. (normal for 5 weeks old infant is 13 gm.) and measured 7 x 5 x 2½ cm. The capsule was smooth. The cut surface showed prominent Malpighian bodies, and the tissue was dark red and firm. On microscopic examination the sinuses were enlarged and filled with erythrocytes. The endothelial cells lining the sinuses were very prominent. The Malpighian bodies were not abnormal. The pulp showed scattered green particles of pigment.

Comment: In congenital spherocytic hemolytic anemia there are probably several factors involved, but the exact role of each is still not entirely clear. Briefly, there is probably an inherited intrinsic red cell defect,[2] which may be due to an autohemolysin, plus a hyperirritability of the spleen,[1] so that exaggeration of a usual hyperhemolysis of the abnormal cell may occur at times to produce a crisis. Owren,[9] however, has presented evidence to suggest that the bone marrow undergoes episodes of temporary aplasia, with the abnormal red cell being destroyed more rapidly by a normally functioning spleen when the bone marrow is unable to meet the demand for increased cells in the peripheral circulation.

The occurrence of hemolytic anemia in infancy is rare, but when crisis occurs it is usually quite severe. Abt[10] recently reviewed the literature on the condition as it occurs in infancy and pointed out the essential clinical and laboratory data.

In cases of this type where the spleen is either over-active *per se* or only over-actively destroying an abnormal cell at rates greater than the marrow can supply them, splenectomy is almost always of marked benefit. The time for carrying out splenectomy in these cases has been somewhat obscure. It is felt by some that splenectomy should be done as soon as the diagnosis is made, without transfusions until after the spleen is out because of the feared danger of intravascular hemolysis and possible kidney damage. This may well be the treatment of choice where the operability of the patient at the time of admission is good. Others feel,[10,12] that transfusions should be given in order to get the patient in optimal operative condition, especially since many patients at this age have a severe crisis and may present a moribund state. In view of this infant's condition on admission, it was felt that it could not withstand anesthesia and operation. Therefore, this procedure was delayed until after transfusions had been given. The response to these was quite good, and the delay in this case no doubt helped decide the favorable outcome.

SUMMARY

Three cases showing hypersplenism in the pediatric age are presented. Two of these made complete recoveries following splenectomy, while the third died immediately.

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Medical Society of Virginia Cancer Committee

Chairman, GEORGE COOPER, JR., M. D.

Medical School Building, University, Virginia

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June 1, 1951

Case Reports

A 54 year old woman who had been conscious of progressive deafness for about two years sought the advice of an otolaryngologist. He inflated the eustachian tubes at periodic intervals for the next seven months. Each time the instrument was introduced into the left tube, there was some bleeding. The deafness continued to progress. The patient noticed and called the attention of the physician to a hard mass in the left mid-anterior cervical chain. At the end of the seven months, the mass was about 6 cm. in diameter and two smaller ones had developed just below it. At this point, the patient visited another doctor who immediately discovered a large tumor in the left nasopharynx invading the eustachian tube and posterior nares. A biopsy from the growth was reported squamous cell carcinoma, grade II. As surgery was considered impractical, palliative radiation was given with good response but the prognosis is hopeless.

Comment: Again we have illustrated the three most common causes of physician responsibility for delayed recognition of cancer:

a. Failure to think of cancer as a diagnostic possibility.
b. Treatment of a condition, the etiology of which has not been determined.
c. Failure to reconsider an initial opinion when the response to treatment is not prompt. Certainly, the bleeding each time an instrument was introduced into the left eustachian tube should have caused the first physician to inspect that area with unusual care, a procedure which would inevitably have led to a much earlier recognition of the correct diagnosis.

In contrast, a 65 year old man was seen recently by another otolaryngologist for routine checkup on the fifth anniversary of a laryngofissure for a small carcinoma of the right cord. The examination was negative. Five years before, the patient had sought advice for hoarseness which had persisted for six weeks after a head cold. With the help of a laryngeal mirror, the physician had discovered the small tumor on that first visit. A biopsy was taken that day, carcinoma was reported, and the laryngofissure was performed four days later.

Cancer **can** be discovered early; it **can** be cured.

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HEMOLYTIC ANEMIA FOLLOWING CAESAREAN SECTION TREATED BY SPLENECTOMY*

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and
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A careful review of the literature has failed to reveal a single case of an acute acquired hemolytic anemia which followed a Caesarean section, performed for a placenta praevia, which was treated successfully by splenectomy. Although acute acquired hemolytic anemia is not common, it occurs with sufficient frequency to be encountered sooner or later by most physicians in the course of an average practice. Virtually all textbooks of Surgery, medicine, pathology and hematology now include some discussion of this subject. In spite of an increasing interest, along with the consequent vast amount of literature, the condition is not as yet fully understood and its clinical management is not as yet standardized.

CASE REPORT

Patient was a 34 year old white female, grava VI, para V, with previous pregnancies in 1942, 1944, 1945, 1946, and 1948.

First seen in the office on September 1, 1949, with history of a L.M.P. on December 25, 1948, and whose E.D.C. was October 2, 1949. All previous pregnancies, except the first in which the baby was born dead, had been normal, with short labors. Examination at first showed no apparent abnormalities. R.B.C. 3,870,000; Hb. 78 per cent; Rh positive; Wassermann, negative.

Admitted to Alexandria Hospital on September 11, 1949, with marked genital bleeding, without any contractions, which had begun that morning at 2:30 A.M. Bleeding ceased and she was allowed to leave the hospital on September 13, 1949.

Readmitted on September 27, 1949, at 4:15 A.M., with moderate bright red genital bleeding and again without contractions. Stat hemogram showed a Hb. of 6 gms. (40 per cent) with a hematocrit of 21. Patient Rh positive, group 0. Given 500 cc. of blood, followed by profuse bleeding. A second 500 cc. of blood was started and the operating room alerted. Examination there under anesthesia showed presence of a central placenta praevia, so an elective Caesarean section was performed with delivery of a normal 7 pound male infant. Patient received five additional pints of blood during and immediately following the operation.

The patient stood the operative procedure remarkably well with the B.P. and vital signs remaining stable throughout. Postoperative course was uneventful during the ensuing few days other than an elevation of the rectal temperature to 101 degrees on the first postoperative day. On October 4th the patient was given 500 cc. of blood as she appeared a bit wan and pale. Twelve hours following this transfusion it was noted that the urine was extremely dark in color and that the temperature was elevated to 101.6. Hb. was 33 per cent with five grams of hemoglobin. On October 6th temperature was still elevated, skin jaundiced, spleen palpable, urine dark in color. Hb. 26.5 per cent with a hematocrit of 11. R.B.C. Fragility test reported as normal with no evidence of spherocytosis on the smear. Given another transfusion and developed a marked reaction one hour after its completion. On October 7th Coomb's test was reported as negative. On October 8th hematocrit had dropped to 9, and reticulocyte count was 6.1. After thorough cross matching the patient was given a transfusion with one of the consulting hematologists as the donor. Developed a severe reaction after 150 cc. of the blood had been given. On October 9th had marked pallor, liver at level of iliac crest, spleen 4 cm. below the costal margin and the hematocrit was 10.

A splenectomy was performed on October 10th. After the pedicle had been clamped the patient was given fifteen hundred cc. of blood and experienced no reaction to the transfusions.

On October 11th hematocrit was 24 and, although it fluctuated up and down during the next three weeks, it still made a steadily upward course. As patient had improved so much, she was allowed to go home on November 1st, 1949, with a hematocrit of 33 and a Hb. of 66 per cent.

*Read before the annual meeting of the Medical Society of Virginia at Roanoke, October 8-11, 1950.

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Since discharge from hospital patient has done very well in spite of the arduous housekeeping duties associated with a household of five small children on a limited income. Repeated checks of the hematocrit and hemoglobin have been performed and have been considered to be satisfactory with no evidence of any continued hemolysis. A complete blood study performed on September 20, 1950, showed the following: Hemoglobin 76 per cent with 11.5 grams; hematocrit 36; blood platelets 220,000; Coomb's negative; no evidence of any spherocytosis in the blood smear.

DISCUSSION

An extensive discussion of the theories of pathogenesis, and mechanism of hemolysis in hemolytic anemias is, of necessity, beyond the scope of this clinical report.

After you have eliminated the possibility of a hemolytic anemia being inherited, or secondary to some recognizable cause, there still remains a group of persons in whom anemia is obviously due to abnormal destruction of red blood cells. In some of these cases, endogenous hemolysins in high titer have been demonstrated in the patient's blood. It is not altogether clear whether isohemolysins are the sole cause of anemia in these patients, or whether these hemolysins are generated by an initial hemolytic episode and merely serve to augment and continue the process.

Some cases of acquired hemolytic anemia manifest themselves with a history of infection, usually a respiratory type infection, a week or ten days prior to the development of the acute symptoms. Many writers have assumed the hemolytic anemia to be the result of the infection, but no etiological agent has as yet been established. In many cases no diligent or thorough search for the serum hemolysins has been made.

Boorman and his coworkers in 1946 demonstrated the existence of adsorbed antibodies not belonging to the Rh-Hr system in cases of acquired hemolytic anemia. By means of a co-called Coomb's or developing test two different types of spherocytes could be distinguished: (1) the uncoated spherocyte usually found in hereditary hemolytic anemia, and (2) the globulin coated spherocyte present in the acquired disease. The developing test should become a necessary requirement in diagnosing hemolytic syndromes. If positive, the hemolytic anemia should be designated as being of the immunologic type. Such an immunologic type may even be found superimposed on hereditary spherocytosis.

Cause of hemolysis is not definitely known. One school believes that the spleen is site of the origin of an antigen and that the whole process is an example of an immune body mechanism similar to mismatched transfusions; the other school feels that the enlarged spleen simply has a greater area in which to perform its usual "graveyard functions." It is therefore entirely possible that the mechanism of causing rapid destruction of red blood cells is associated with sensitization in the reticulo-endothelial system.

The spleen is occasionally more of a hindrance than a help, and certain forms of disordered splenic function are revealed by the accelerated destruction of red blood cells, or by a pathological influence on blood cell formation. The beneficial and often curative effect of splenectomy in a significant number of cases is ample proof that the spleen is the principal offender; the means by which the organ does harm is not so well understood. The term *hypersplenism* is recommended to cover this particular group of splenic disorders, which are based on harmful or excessive splenic function. Hypersplenism limited to red blood cells is exemplified by familial hemolytic jaundice, where splenectomy nearly always effects a clinical cure. In the less specific forms of acquired hemolytic anemia there is a lower incidence of clinical cure indicating that the spleen is less often the site of the disturbance.

When patient continues to have hemolysis after several transfusions, it is the consensus of opinion that splenectomy should be performed and that one should not wait too long before resorting to this procedure. There is a tendency among some medical men to feel that the patient will not withstand the operation, but it should be recognized that without splenectomy the outcome is, or may be, hopeless.

It is readily acknowledged that each case must be treated individually and the old aphorism is still true—the exception proves the rule—that every case may not show the accepted or pathognomonic findings.

It is entirely within the realm of possibility that two or more entirely different factors or causes may have been present in this case.

Development of increasingly more severe reactions

after repeated transfusions might suggest some isoimmunization either with agglutinins or hemolysins, though none could be demonstrated. Transfused normal cells become involved in the hemolytic process and are eliminated from the circulation at an accelerated rate and may well have occurred in this particular case.

Bone marrow biopsy is of negative value in an acute hemolytic anemia as it shows only a normoblastic hyperplasia and in that way rules out other types of blood disorders.

Hydrogen ion concentration of the serum is thought to have an influence on the hemolytic system, with hemolysis within certain ranges found to be more frequent.

In this case a thorough search for the cause of the sudden onset of, and continued hemolysis was made without success. Careful and close check of the blood to be transfused, as well as the serum of the patient, to rule out the possibility of some incompatibility was also performed without success.

To all intents and purposes the condition which this patient experienced was like a "bolt out of the blue," and was that of an acute acquired idiopathic hemolytic anemia. The cause of the hemolysis was never determined and at no time could be attributed to any drug, toxic substance or metabolic disturbance, although the possibility of one or more of these factors being the cause remains quite likely.

The decision to perform the splenectomy was made after thorough hematologic study had failed to reveal the cause of the hemolysis and when the condition of the patient was steadily becoming more critical hour by hour. The results of the operation were most satisfying, and tend to bear out the thoughts of so many men who recommend a splenectomy in cases of acquired hemolytic anemia in which the patient fails to respond to repeated transfusions, and this decision should be made before the condition of the patient becomes too critical for such an operation.

The follow-up on this patient as mentioned in the Case Report has shown a return of the hematocrit and hemoglobin to acceptable levels and there has been no evidence of any repeated hemolysis since the patient's discharge from the hospital almost one year ago.

SUMMARY

We have presented the case of a multiparous young white female who, following a Caesarean section for placenta praevia, developed an acute acquired idiopathic hemolytic anemia. The etiology of the anemia was never determined, in spite of an extensive hematological workup. Splenectomy was performed after the patient continued to show hemolysis and continued to go down hill, in spite of repeated transfusions together with extensive symptomatic and supportive therapy. Patient made a very satisfactory recovery from the operation and has not shown any hemolysis since her discharge from the hospital.

In addition, we have discussed some of the more current ideas as to the cause of acquired hemolytic anemias and have presented the thought that splenectomy should always be kept in mind in case the patient does not respond to repeated transfusions and conservative management. The decision to perform splenectomy should be made before the condition of the patient becomes so critical that the operation would not be practical.

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DISCUSSION

DR. HERBERT H. SCHOENFELD, Washington, D. C.: Acute anemias are frequently spectacular in their picture as well as dramatic in their relief.

Heroic surgery, when indicated, as in the report of Dr. Delaney, is certainly to be commended.

It is my judgment that, with the still present moot of splenic physiology, we should be more frequently radical in our treatment of hemolytic anemias, regardless of cause, even when such cause is undetermined, unless a quick reverse of the pathological progress of hemolysis is attained by other therapy.

Certainly, in a patient whose hemolytic process is progressive, and in whom no adequate cause can be determined, and in whom palpable splenomegaly and hepatomegaly occurs, we should grasp the only real definite treatment which might avail.

It is to be noted also that apparent splenomegaly may not be present in all cases.

DR. C. L. RILEY, Winchester: The speaker is to be congratulated upon the presentation of a very interesting paper, and the splendid results obtained. More and more attention is being given to blood dyscrasias associated with pregnancy, particularly those in which splenectomy is indicated. The importance of the proper timing of operation in puerpera hemorrhagica, hemolytic anemias, etc., should be emphasized in that acute exacerbations do arise during the puerperium and death has occurred from uncontrollable hemorrhages during this period.

A patient near term was recently seen with puerpera hemorrhagica in which a precipitous fall in platelet count occurred and hemorrhagic tendencies developed so rapidly that active treatment was considered imperative. Splenectomy and cesarean section (the former by Dr. L. W. Woodward, Woodstock, Va.) were done simultaneously with gratifying results.

The author's case represents one of the few of an acquired hemolytic anemia during or following pregnancy that has been reported and illustrates well the necessity for prompt and adequate therapy.

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TREATMENT OF EPILEPSY—A STUDY OF ELEVEN CONSECUTIVE CASES*

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Rapid and revolutionary advances have been made in the study and treatment of epilepsy during the past few years. As a result it is apparent that nearly all patients who are capable of carrying out proper instructions can live without seizures. Indeed, for the past eighteen months the writer has been assuring nearly every patient with epilepsy that his seizures can be stopped. Such assurance has tremendous therapeutic value. Of course, this assurance is given only after many hours of studying the patient and after ruling out any appreciable possibility of a progressive intracranial lesion.

The writer believes a report of his more recent experiences in the study and treatment of epileptics may be of value to other practitioners. He offers 11 consecutive case studies.

Case 1: White male, age 30. When first seen in August, 1948, this patient was having grand mal seizures about five times a month, usually with aura, and petit mal attacks six or seven times a day. Two or three times a month he was having psychomotor attacks consisting of about ½ hour of amnesia, usually with running fits. He then was taking regularly mesantoin gr. 1-½, t.i.d. He previously had done no better on dilantin gr. 1-½, t.i.d., with phenobarbital gr. ½, t.i.d. He was unemployed and unemployable, and discouraged by his parents from attempts at employment lest he lose his pension. He spent his time brooding over bitter childhood experiences and bitter war experiences. Seizures developed in 1946, two years after hospital treatment for filariasis.

The only positive physical or neurological findings were absence of swing of right arm and a vague drag of right foot evidenced only by comparison of his shoe soles. Electroencephalogram in January, 1948, showed only normal variation.

At the first appointment he was given a two-hour interview of study and psychotherapy. This was followed during the next few weeks by many one-hour conferences. Much attention was given to ameliorating his disturbing emotions, improving the home atmosphere, getting him to seek the fellowship of others, and helping him to work out ways of replacing the time spent in bitterness with all types of activities suitable to an epileptic. At the end of the intensive study he was given absolute assurance that with his cooperation his seizures could be stopped by proper combinations of the drugs now available, used in adequate doses. He was told that the doses would be increased gradually and that perhaps they would make him temporarily sick; but if one drug or group of drugs made him sick, there was a great variety of others which would not. He was instructed to telephone at once for an appointment if a rash, sore gums, dizziness, indigestion, or any new complaints of any kind developed, even if it seemed to be no more than a "fresh cold", as the possibility of its being caused by one of the drugs must be ascertained at once. In order to avoid errors of overcaution in medication, patient was instructed that if it seemed necessary to see another physician or dentist, such consultation should be arranged by the physician treating his major condition, epilepsy. He was instructed to telephone promptly if another seizure occurred. He was instructed to clean his teeth gently after every meal and at bedtime.

Patient was told that doctors have produced seizures for diagnostic purposes by having a patient drink excessive amounts of fluid after a dose of pitressin. He was instructed to drink the least amount of fluids consistent with comfort, never a tumbler full if a tumbler half full would quench his thirst.

Patient was told that doctors also have produced seizures for diagnostic purposes by having a patient breathe excessively for a few minutes. He was reminded that anger, bitterness, fear, worry, and kindred emotional brain storms produce such excessive breathing, and was told that in the writer's opinion this over-ventilation is of major importance in bringing on each seizure. He was urged, therefore, to use every possible means of preventing such brain storms and to "put on his thinking cap" in order to work out better past-times. He also was instructed that if or when he ever should find himself in a

*Read before the Seaboard Medical Association of Virginia and North Carolina, December 6, 1949.

tense emotional state, he was at once to start breathing very slowly and to take in the least amount of air compatible with comfort. He was told that this often would prevent the occurrence of seizures, and it also would reduce greatly the emotional tenseness itself.

Patient was instructed to eat a small snack between meals and a dry snack at bedtime as, in the writer's experience, this reduces tendencies to emotional tenseness and to seizures of all types.

Patient was instructed never to exert himself physically more than 10 per cent beyond his usual activities because such over-exertion sometimes brings on seizures. He was advised, however, to increase gradually his physical activities because habitual physical activity seems to reduce the tendency to seizures.

Patient was advised and urged to start making plans for life employment in some occupation considered safe for an epileptic.

Medication was changed to mesantoin gr. 1-½, b.i.d., dilantin gr. 1-½, b.i.d., and phenobarbital gr. 1-½ at bedtime.

The grand mal seizures ceased, and the petit mal and psychomotor attacks became very infrequent and mild. He applied for a course of training under the Veterans Administration Rehabilitation Program, and the anticipation of this further improved the patient.

There followed many months of waiting for the course to be started until patient grew so impatient that he impulsively made a trip to Washington hoping to induce his senator to get him back into the army despite his epileptic history. He was disappointed and embittered over not being permitted to see the senator, and had three grand mal seizures during the following 48 hours. This episode occurred in January, 1949.

Medication was increased to dilantin gr. 1-½, t.i.d., mesantoin gr. 1-½, t.i.d., and phenobarbital gr. 1-½ at bedtime. About three weeks later he was started on his course in radio repair work which he has continued regularly and zealously. He has made good grades and has been very happy and well adjusted in every way. Since January 16, 1949, eleven months ago, there have been no grand mal or petit mal seizures, no psychomotor attacks, and no physical or nervous complaints of any kind.

Case 2: Colored male, age 33. When first seen February 2, 1949, patient was having grand mal seizures, usually twice a month, and usually with dislocation of his jaw. He had had seizures for eight years. He had been entirely unemployed for 1-½ years, and was unemployable, very tense, and very unhappy. He had had syphilis, possibly congenital, and this may have been a major factor in his epilepsy. About the time of onset of seizures he was having bilateral keratitis which left him with corrected vision in his best eye of 20/100. When first seen, repeated blood serology had been negative for syphilis for six years. A spinal fluid was obtained and found negative. Physical and neurological examinations were negative for any evidence or residuals of syphilis other than the damaged eyes.

Patient was given the same long and thorough study as was given to *Case 1,* the same psychotherapy, the same assurance that seizures could and would be stopped, the same extensive instructions including those referable to possible factors that might cause or that might prevent seizures. He was given dilantin gr. 1-½, t.i.d., with phenobarbital gr. 1-½, at bedtime. In April, two months after starting treatment, while attending very exciting religious services, he had two seizures within 24 hours. He had one seizure in June because of the same excitement. On each of these two occasions his daily dilantin was increased gr. 1-½. Since June he has been taking 7-½ gr. dilantin a day and phenobarbital gr. 1-½, at bedtime. On this medication for six months there have been no further seizures and no physical complaints of any kind other than the defective vision mentioned above. Since September 15, he has been employed regularly at suitable work.

Case 3: Colored female, age 25. When first seen, July 7, 1949, patient was having grand mal seizures once or twice a month, often with minor injuries. She was unemployed and unemployable, dependent upon relatives, quarrelsome, tense, and unhappy. Her childhood had been made unhappy by a brutal father. Onset was at the age of 13 years, one month after a fall which raised a bump on her head but did not produce unconsciousness nor sequelae. A brother who died, age 16, had had seizures following a head injury. Her mother emphasized the fact that ever since early childhood patient had been sensitive, bashful, and withdrawn.

Patient was given the same thorough study, psychotherapy, and instructions, and assurance as were

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Cases 1 and *2.* Treatment included dilantin gr. 1-½, t.i.d., with phenobarbital gr. 1-½, at bedtime. There occurred a rash and high fever, believed due to dilantin. Medication was changed slowly to phenobarbital gr. 3, at bedtime, and gr. ½, b.i.d.

There have been no further seizures, nor other symptoms of epilepsy during the five months since the beginning of treatment. Patient is working half-time, is practically self-supporting, and appears very happy.

Case 4: White girl, age 13. When first seen, May 2, 1949, patient was in status epilepticus and was wearing a "Statue of Liberty" cast for fracture of the left humerus. There had been 10 seizures within a few hours, but seizures promptly subsided after liberal doses of phenobarbital. Prior to status epilepticus she had been having a seizure about every two days. Patient also had had psychomotor attacks and petit mal attacks. Onset was at age two. There had been maladjustment throughout her life.

Patient had been studied and treated several times at a medical college hospital and her seizures stopped; but each time parents would discontinue medicines and seizures would return.

Physical and neurological examinations were negative except for: the fracture, a small shiny raised body lateral to the disc of right eye (not believed etiologically significant), and a rash said to be present since tridione was discontinued three months previously. X-ray showed non-union of the fracture. Intelligence is subnormal, I.Q. being 60.

Treatment included the same thorough study and psychotherapy as were given to *Case 1.* The same instructions and assurance were given to the child and to the parents. Because of the fracture, cessation of seizures seemed imperative; and she was at once put on dilantin gr. 1-½, four times a day and phenobarbital gr. 1-½, at bedtime. Five months later gums became swollen, and dilantin was reduced to gr. 1-½, t.i.d.

There have been no further grand mal seizures since patient was first seen. There have been periods of mental dullness, lasting a few hours, which may have been psychomotor attacks, but more likely were periods of schizoid behavior. It was learned that she was being teased and tormented by schoolmates. This was stopped by sending her to another school. Since then the dull periods have ceased and her school work has improved.

Case 5: White female, age 25. When first seen August 2, 1949, patient was in status epilepticus which promptly subsided on liberal doses of phenobarbital. Although taking dilantin gr. 1-½, four times a day she had been having about eight grand mal seizures a month and also some petit mal and psychomotor attacks. She had been having seizures since seven months of age and had spent two periods of about two months each in a state hospital. Aura consisted of stomach pain and nervousness. An aunt and a cousin were epileptic. Because of seizures and because of patient's unpredictable temperament, it was considered necessary for her mother-in-law to run the home, a situation which patient bitterly resented.

Treatment included the same thorough study, psychotherapy, instructions, and assurance described above; and a slow change in medication to dilantin gr. 1-½, t.i.d., mesantoin gr. 1-½, t.i.d., and phenobarbital gr. 1-½ at bedtime.

During the four months since first seen there have been three attacks of petit mal and two grand mal seizures. The first grand mal seizure occurred while patient's aged father-in-law had pneumonia and patient was tensely over-working. The second seizure occurred November 14, 1949, following changes in medication while she had an infection of mouth that was growing progressively worse. She now is taking dilantin gr. 1-½, t.i.d., mesantoin gr. 1-½, four times a day, phenobarbital gr. 2 at bedtime, and gr. ½, b.i.d. During the four months she has grown fairly happy in her outlook, has run her home fairly well for 1-½ months in the absence of her mother-in-law, and seems free of any direct effects of her epilepsy. However, it must be admitted that she shows considerable evidence of a schizoid personality that is made more manifest by her experiences as an epileptic. This evidence consists of childish quarrelsomeness, irritability, and undue sensitiveness resulting in shut-in habits.

Case 6: White girl, age 8. When first seen, April, 1947, patient was having 6 to 12 petit mal attacks a day, onset being five months previous. The pattern fitted that described as pyknolepsy. The attacks lasted only a few seconds and never resulted in a fall. Mother was very religious, strict, and of a rigid personality. Patient was thin and of "race horse" habitus and habits, in which she resembled her father.

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Treatment included the same thorough study, psychotherapy, and the same instructions and assurance to patient and her family described above. Special emphasis was placed upon frequent feedings, as children of her "race horse" build seem prone to a relative hypoglycemia with resulting symptoms. Tridione was started and attacks became infrequent. It later was increased to gr. 5, four times a day. Within another two months all of the attacks ceased and there has been no return, although the tridione was withdrawn slowly over a period of four months.

Case 7: White male, age 23. When first seen April, 1949, patient was having a grand mal seizure every few months usually so severe as to necessitate his losing two days from work. Onset was four years ago. Aura consisted of a feeling of tenseness and a numbness over right half of face. Often he would stay home from work because of tenseness and fear of a seizure, his occupation being hazardous for an epileptic. All seizures occurred at times of emotional stress or following unusual physical fatigue. Patient states his first two seizures occurred two weeks after a fall in 1945, while in military service. Six months later there was a craniotomy. Examination showed right arm smaller and weaker than the left, and a narrowing of field of vision of right eye. Possibly these defects dated from a birth injury he is known to have had. EEG made in June 1949, was "suggestive of epilepsy".

Treatment included the same thorough study, psychotherapy, instructions, and assurance described above, successful efforts in getting patient to secure safe employment, and a change in medication from dilantin gr. 1-½, t.i.d., with phenobarbital gr. 1-½, at bedtime, to dilantin gr. 1-½, and mesantoin gr. 1-½, each b.i.d., with phenobarbital gr. 1-½ at bedtime.

During the eight months of treatment there have been no further seizures and no auras.

Case 8: Colored male, age 23. This atypical or questionable case, when first seen in March, 1948, carried a diagnosis of idiopathic epilepsy. Patient's spells consisted of syncope following emotional upsets or following over-exertion. These spells were followed by headache and weakness which, prior to treatment, resulted in the loss of much time from his work as a college student. His build is thin, the "race horse" type. He always carries marked physical evidence of a stimulated sympathetic nervous system. In the past he had been found to have a flat glucose tolerance curve and fasting blood sugar as low as 40. Numerous studies including three EEG's during the past three years have left it questionable whether this is a true case of epilepsy.

Treatment has included much psychotherapy. Medication prescribed was dilantin gr. 1-½, t.i.d., with phenobarbital gr. 1-½ at bedtime. Treatment has decreased the evidence of nervous tension only slightly, and somewhat reduced the number of spells. However, the spells are much milder and rarely keep him from work; and the quality of his college work has definitely improved. This is perhaps a case of anxiety neurosis with functional hypoglycemia resulting in attacks of syncope.

Case 9: Colored female, age 45. When first seen in August, 1948, patient was in status epilepticus which promptly subsided following the administration of adequate phenobarbital. Since age five she had had frequent grand mal seizures and psychomotor attacks. She was given psychotherapy and started on dilantin gr. 1-½ in the morning and gr. 3 at bedtime. Except for one period in March, 1949, when she ceased taking her medicine, there have been no further seizures.

Case 10: White female, age 28. When first seen January, 1948, patient was having as many as four seizures a month. She was given psychotherapy and started on dilantin gr. 1-½, b.i.d., with phenobarbital gr. 1 at bedtime. She improved, but has had a seizure every few months. Upon each of these occasions the seizure was preceded by an emotional upset with resulting failure to take medicine.

Case 11: White female, age 40. When first seen 12-22-48, patient was in a hospital, confused, and exhibiting bizarre, senseless, excited behavior which the referring physician called hysteria but which the writer considered to be a psychomotor attack. For four years she had had frequent similar attacks, some of them with possible grand mal, usually lasting a few hours to a few days. Many of these spells were preceded by alcoholism. All were preceded by some occasion of an emotional upset.

Early history included desertion by father in early childhood and the death of her mother when patient was age 19. She was too nervous to work the following year. She retains bitterness toward her sister referable to occurrences preceding her mother's death.

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Physical examination was essentially negative except for hirsutism.

Electroencephalogram showed dysrhythmic record consistent with epilepsy, suggestive of psychomotor pattern in right occiput. X-ray of the skull was negative.

Treatment included psychotherapy. Medication was soon built up to dilantin gr. 1-½, four times a day, with phenobarbital gr. 1-½ at bedtime. Following her third spell after initiation of treatment, medication was finally changed to dilantin gr. 1-½, t.i.d., mesantoin gr. 1-1½, b.i.d., and phenobarbital gr. 1-½, at bedtime. She was questioned closely following each of these three spells, and it was learned that each was preceded by the occasion of an emotional upset. Each was judged to be a psychomotor seizure and of the type that many doctors call "hysteria". Since the spell and change in medication, 4-15-50, there has been only one spell. This one followed the illness and death of a member of her family at which time she had neglected her medication. All of these four spells were relatively mild and brief, and did not require hospitalization as did many of the former ones.

From experience and from a review of the more recent reports, certain conclusions are presented:

The most effective drugs for grand mal seizures are phenobarbital and dilantin. Dilantin also often decreases psychomotor attacks. When seizures cannot be controlled satisfactorily by dilantin and phenobarbital, they often can be by adding mesantoin. With either dilantin or mesantoin much better results can be obtained if a bedtime dose of phenobarbital gr. 1-½, is given. The writer nearly always does this, but rarely gives phenobarbital in the daytime as the resulting drowsiness discourages the patient. Still better results often can be obtained if all three of these drugs are given.

A very common error in medication is inadequate dosage. The amount necessary of dilantin and of mesantoin often reaches the toxic level. Time will not be taken here to review the numerous toxic effects of these drugs; but doctors cannot properly treat epilepsy without giving their patients an understanding of the drugs used and their toxic effects. Frequent interviews are necessary.

In starting dilantin or mesantoin, safety demands that the dose be built up gradually. Mesantoin is started at one dose a day of gr. 1-½; dilantin at two such doses. If seizures persist, an additional daily dose is added every few days. If three or four such doses a day of either drug does not give satisfactory results, the writer usually starts the other drug also, slowly building up the dose. Each case must be individualized. The writer is giving many of his patients the following combination: dilantin gr. 1-½, t.i.d., mesantoin gr. 1-½, t.i.d., and phenobarbital gr. 1-½ at bedtime.

The above drugs seldom have beneficial effect upon petit mal. This can be controlled and often stopped by tridione in adequate doses. It has produced fatal blood dyscrasia and often has other toxic results, especially in adults, and more especially in those who have reached middle life. It should not be given to arteriosclerotics for spells resembling petit mal. In children and in younger adults with both petit mal and grand mal it usually is satisfactory to combine tridione with dilantin and bedtime phenobarbital. A high portion of the reported cases of agranulocytosis occurred while patients were taking both tridione and mesantoin.

There are other promising drugs. Meboral, a barbituate, appears to produce less depression of intellectual functions than phenobarbital. Thiantoin sodium (phethenylate sodium) has been offered as being more effective in proportion to its toxicity than dilantin or mesantoin. Similarly, paradione has been offered as a substitute for tridione. Dextro-amphetamine (dexedrine) has been used to counteract the sedative effects of phenobarbital and other drugs. It is helpful for petit mal and psychomotor attacks and for behavior disorders even when apparently not the result of epilepsy.

The writer's experience has confirmed his deduction that seizures sometimes can be prevented by slow breathing. Therefore, he carefully explains to each patient that over-ventilation, incident to emotional upsets, can cause seizures and should be avoided by slow breathing. He also explains the importance of drinking no more fluid than is necessary for comfort. His only routine instruction as to diet is that excess hunger must be carefully avoided by eating a snack between meals and at bedtime, although he can offer no precedent for routinely doing this. However, some workers report that hypoglycemia can produce seizures of both grand mal and petit mal types; other workers say that it sometimes enters into the mechanisms of the production of the physical complaints produced by anxiety reactions and by a great

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variety of psychosomatic diseases. The writer's experience confirms this.

Many workers emphasize the importance of emotional upsets in producing the individual seizures. When a patient is taking regularly substantial doses of suitable drugs but still is having occasional seizures, the writer has observed that on practically every occasion the precipitating cause is an emotional upset, and that this upset is nearly always of a type which the patient can be taught to prevent, or at least to soften.

From experience the writer is convinced that fear of seizures is a major cause of seizures, and in the great majority of cases he assures the patient that if all directions are followed, his seizures will stop—thus taking a well calculated and relatively insignificant risk to his reputation.

Long experience in carefully taking histories has led the writer to question the conventional view that epilepsy is primarily genetic rather than environmental. Of course, a constitutional diathesis, that is, a genetically determined low threshold for seizures does exist. The high incidence of EEG dysrhythmia in relatives of epileptics (60 per cent) is considered proof that epilepsy is genetically determined. However, no studies appear available to prove that life experiences and environment (an unhappy or a tense home) do not similarly influence the EEG rhythm. We do not know why only one out of twenty people with dysrhythmia have seizures. We do not know as yet whether a genetic tendency and an unhappy tense environment are both necessary to produce epilepsy in the absence of intracranial lesions. Neither do we know whether the mechanism of producing seizures involves psychic over-production or psychic under-production of some endocrine or hormone, perhaps as yet unknown or inadequately studied.

In the present state of our ignorance and groping, the writer takes a careful history of all emotional factors likely to produce the epileptic constitution or likely to cause the individual seizure. The present environment is reviewed including the whole family, and the patient's school or his employment. Changes are attempted whenever indicated and practical. The patient's habits are studied especially, and improvement in mental hygiene is attempted. If patient is unemployed, he is induced to make plans for early employment. Attempts are made to lessen emotional reactions by psychotherapy. All of this is done as early as possible. In general, the writer includes in his treatment of epileptics all of the psychotherapeutic measures he uses for neurotics, and for patients with psychosomatic diseases.

It is the writer's opinion, based upon his experience, that if every available means is used consistently, nearly all epileptics can live without seizures. To this end, the writer wishes to emphasize the fact that all eleven of these consecutive cases now (Nov. 25, 1949) are adjusting as normal individuals, all living purposefully as industrial workers, as students, or as satisfactory housewives.

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ACTH Helps Severely Burned Children.

Further evidence that ACTH is a valuable aid in the treatment of severely burned persons was reported by four Minneapolis doctors in the May 5th *Journal of the American Medical Association.*

Drs. Forrest H. Adams, Eldon Berglund, Samuel G. Balkin and Tague Chisholm, who administered the drug to three severely burned children, reported that there was a decrease in the fever, an increase in appetite and an improvement in the emotional status of the children. They received the hormone for five, 30 and 100 days respectively. The doctors are associated with the University of Minnesota Medical School and the Minnesota General Hospital.

The doctors explained that fever, lack of appetite, irritability and emotional instability, so often present in severely burned persons, pose many problems to the attending physician. For example, loss of appetite—probably related to the fever—and the loss of body fluids and proteins from the burned area might produce a state of malnutrition, making skin graft difficult. The use of ACTH in severely burned patients is, "a rational form of treatment justifying further investigation."

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CLINICOPATHOLOGICAL REPORTS

From the Case Records of the Medical College of Virginia and the University of Virginia Hospitals.

HARRY WALKER, M.D., *Editor*
WILLIAM KAY, M.D., *Associate Editor*

CASE NO.: 54

The patient, a 67 year old, white, retired railroad worker, was admitted to the hospital on January 1, 1950, complaining of "pain in the stomach and headache." He had been in good health until about two months prior to admission when he had the "flu". Since then he felt weak and rundown. For about a week to ten days prior to admission he had occasional vague abdominal pains, which he located to the right of his umbilicus. The pains were dull in nature and usually occurred at night. Lying down would usually relieve the pain. Vomiting occurred twice during the past month; no blood was seen. He was able to tolerate all foods and was unable to give any reason for the abdominal pain. There has been no change in bowel habit, color of his stools, and no chills or fever. One week before admission he began to have headache which was located in the frontal and occipital areas. It was dull and brought on or aggravated by stooping or moving his head quickly, and not relieved by aspirin. About five days before admission he noticed that his urine was becoming darker. There had been a weight loss of 10 or 15 pounds in the past two months.

The past, family, and marital histories were not remarkable. The review of systems were essentially negative. The patient had had all of his teeth extracted nine months ago. For many year there had been nocturia twice each night. He was a heavy smoker, but drank alcohol only in moderation.

Physical Examination: Temperature 100°F. rectally, respirations 24, pulse 94, blood pressure 100/-65. The patient was a well developed and well nourished male in no apparent distress. He was alert and oriented. The skin over the face was described as ruddy and icteric. The sclerae were also jaundiced. The left pupil was larger than the right and oval in shape. There was a perforation of the right ear drum which appeared to be old. The tongue was negative. The thyroid was not palpable. The thorax was increased in AP diameter, but was symmetrical. The lungs were clear to percussion and auscultation. The heart size was difficult to evaluate because of the large chest. The heart sounds were distant, no murmurs were heard, and the rhythm was regular. The liver was enlarged 2 finger breadths below the costal margin; it was hard and non-tender. There was some moderate tenderness deep in the right upper quadrant, but no masses were felt. The spleen was not felt. No other masses were felt or tenderness noted in the abdomen. The prostate was slightly enlarged. It was smooth and non-tender. The extremities were negative. The reflexes were equal and active.

Laboratory studies on admission: Urine was amber, cloudy, specific gravity 1.020, there was 1+ albumin, 1+ sugar, no acetone but positive for bile. There were 1-2 red cells and 2-4 white cells per h.p.f. The hemoglobin was 10.8 grams with 4,900,-000 red cells, and 13,000 white cells. There were 80% polys., 17% lymphs., and 3% eosinophils. The serology was negative. Cephalin flocculation 4+, total proteins 7.3 with 2.5 albumin and 4.8 globulin, serum bilirubin direct 2.7, mgs. total 4.0 mgs. The BSP was 36.8%. Serum amylase was 19 units; repeated blood cultures were negative. One stool examination showed no amoeba.

The patient became progressively more jaundiced and ran a spiking type temperature ranging from normal to 102°F. Repeat liver function test one week after admission showed a cephalin flocculation of 4+, 7.0 total proteins, 2.5 albumin and 4.5 globulin. The serum bilirubin direct was 2.8 mgs.% and total was 4.0 mgs.%. BSP was 36.8%. Examination of the stool was positive for urobilin. Blood cultures continued to be negative. A gastro-intestinal series was not remarkable except for a duodenal diverticulum. The patient continued to complain of weakness and vague upper abdominal discomfort.

An exploratory laparotomy was performed and the liver was found to be enlarged three finger breadths below the right costal margin. The margins of the liver were rounded and the organ was slate gray in color. The surface was irregular due to numerous

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nodules. A biopsy taken from one of the larger nodules showed "hepatitis and fibrosis" of the liver substance. The gall bladder appeared to be of normal size and consistency, and no stones could be palpated in the gall bladder or common duct. The remainder of the upper abdomen appeared normal. A small T tube was inserted in the common duct; a cigarette drain was left in the wound.

Following the operation the patient seemed to improve, the wound drained bile freely, his temperature became lower and his general condition appeared better. One week following the operation the drain was removed and ascitic fluid seeped from the wound. During this time the patient was afebrile. He later began to gradually become progessively weaker. He was put on a high protein, high caloric diet which he tolerated well. His hemoglobin at this time was 10.8 gms. with 4,200,000 red cells and 15,000 white cells. The NPN was 42 mgs.%, the serum bilirubin direct was 5.4 mgm., a total of 8.4 mgm.%. Cephalin flocculation was 2+. Total protein was 7.5 with 2.4 grams of albumin, 5.1 of globulin. On the 11the post-operative day the patient began running a low-grade fever which continued in spite of antibiotics. The jaundice became progressively worse, the serum bilirubin on the 14th post-operative day was 15.4 mgm.% direct and 22.4 mgm.% total bilirubin. Cephalin flocculation was 1+ and stools were negative for urobilinogen. His temperature began rising and was again septic in nature. The patient became increasingly weaker and became confused and disoriented. The fluid output remained very good but he became more and more drowsy. His wound drained small amounts of bile stained material. The liver edge remained palpable about three finger breadths below the costal margin. The patient became comatose and finally died on the 14th of February, his 30th post-operative day.

Treatment during the entire hospital stay was supportive and symptomatic. Following the operation he was given crysticillin, a protein digest to take by mouth and methionine, 0.2 grams, twice daily. He was also given intramuscular crude liver extract, I. V. glucose and several whole blood transfusions.

DISCUSSION BY DR. CHARLES M. CARAVATI*

This is a 67 year old, white male, who was a railroad worker, who was well until about two months prior to his admission to the hospital, at which time he began to have malaise, or something akin to it, which was described as a possible "flu". Ten days before admission, this patient developed abdominal pains of a rather unusually and bizarre type, which were localized around the umbilicus to the right and which, interestingly enough, disappeared or improved upon reclining. One of the important points about abdominal pain is how it is relieved. In this patient the pain was relieved when lying down which would suggest that the pain may have been previously due to traction on some organ or some mechanical factor, rather than due to any of the usual causes of abdominal pain. I gather that the pain was not too significant—it wasn't too bad, its intensity was not great. A few days later the patient developed a headache of an occipital and frontal type—again the pattern is difficult to make much of; and at the same time, he probably had fever, so one questions the fact whether the headache was significant. Five days before admission, he had dark urine. During this time of two months, he had lost approximately 10 to 15 pounds in weight. His past and his family history and his personal history were not significant and his symptoms review revealed nothing that helps us. He had had a tooth extracted 9 months before. He had had nocturia twice a night for a number of years. His physical examination showed an enlarged prostate without nodules, so that may be the explanation of his nocturia. Relative to his personal habits, it may be significant to note that he drank moderately, whatever that may be, and also smoked heavily.

On physical examination when he came into the hospital, he had a temperature of 100.8°F., and he appeared to be not too sick—he was well developed and well nourished. He had a definite icterus. His left pupil was larger than his right pupil, and it was oval in shape. Whether that is significant or not, I do not know. We are given no other information; one begins to think, of course, of some lesion that may disturb the constriction of the pupil, but we are not told anything about accommodation or about the reaction to light. Therefore, it seems we cannot do anything but discard it because we do not know whether it was just an inequality of pupils or not. He had a perforated ear drum. His chest was essentially negative. His liver was enlarged two finger breadths below the costal margin—it was

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hard, it was irregular and it was non-tender. There was some tenderness below the liver deep in the right upper quadrant. This was not particularly pronounced, but I think it was important because it was localized. There were no other findings in his abdomen of any consequence; his spleen was not palpable. I have already mentioned that his prostate was slightly enlarged.

His course was an interesting one. From the beginning, he manifested a febrile course of a spiking type. The temperature would go down to normal and then spike as high as 102.0°F. This continued until he was operated on. At the same time he had jaundice which was fairly stationary at the beginning but later deepened. Associated with the jaundice were the symptoms of weakness and vague upper abdominal distress. At that time he was x-rayed. The x-ray is of little help because it is negative. We can discard it by saying that the upper alimentary tract is essentially negative for organic pathology from a roentgen viewpoint.

In view of the fact that this man's course continued poor, it was decided to do a laparotomy. I do not know why they thought a laparotomy might be helpful, but I suspect that with the presence of a spiking fever and an enlarged liver, the clinicians felt that the bet was that this patient had a liver abscess. However, they may have been thinking of suppurative cholangiolitis associated with obstruction of the common duct. It would appear, however, that they did not try hard to find out about the presence of cholangiolitis as one gathers later from the laboratory studies and from the study of the stools, so I am not particularly convinced that they were concerned too much about that diagnosis.

It seems to me that they began to wonder whether or not he had an abscess of the liver, either single or multiple. The stool studies for amoebae and other tests which were done would indicate that they may have thought that he had an abscess in his liver. I think the other fact is that they did not know what the difficulty was so they thought an exploratory laparotomy might be helpful. It was; it showed a liver which was slate-gray in color; which was enlarged and on the surface of which were many large nodules. The common bile duct was normal; the gall-bladder was normal in size and contained no stones. There was no dilatation of the common duct, and apparently no obstruction at the sphincter. These findings, of course, are extremely important and helpful. More important still, is that the exploration of the remainder of the upper abdomen was negative, so the conclusion was that the pathology rested in the liver. A biopsy of the liver was taken and a T tube was inserted in the common duct. After the operation, the temperature came down and for ten days was normal, but in the meantime the pathologist sent in a report saying that this represented "hepatitis and fibrosis" of the liver whatever that may mean. However, the patient did not remain well too long, though interestingly enough, he had excellent drainage of bile through his T tube. His temperature came down and he appeared to be somewhat better but soon his temperature went up again, and he began to feel weak and prostrated, his jaundice began to deepen, and he was losing ground consistently. The patient gradually went downhill and died, and there was nothing else unusual noted at this time, particularly as regards change in the size or consistency of his liver and development of ascites. His death was slow and apparently was precipitated by no particular complication.

The patient had a low grade anemia with a hemoglobin of 10.8 grams on two occasions, both when he first came in and again later. The red cell count was approximately 4 million so his anemia was not striking. On two different occasions, he had a mild leukocytosis with a fairly normal spread on the differential—80% polys, 17% lymphs and % eosinophils. Blood chemistry showed a 2+, 4+ and then a 1 + cephalin flocculation and one wonders why the fluctuation in this test. We must remember that the cephalin flocculation is rather susceptible to many changes in one's general condition; namely, too high fever and many other factors, so perhaps this is a nonspecific reading and does not mean that it represents hepatic dysfunction. The BSP was exactly 36.8% on two different occasions. In a man with jaundice, this would mean nothing to me. If I am wrong, I would like Dr. Kuppfer and Dr. Young to comment on it. In the first place, I think all of us agree that it is hazardous to run a BSP in a case of jaundice, except when the laboratory uses a particular photoelectric colorimeter with which they can differentiate the color change in the serum carefully. It is commented that the finest way to get an increased BSP is just to produce fever in anybody—nonspecific fever will very often result in re-

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tention of BSP, though not as high as 36% as a general rule. Investigators at the University of Virginia showed this by giving typhoid fever intravenously. At the height of the fever there was a retention of BSP. This patient had a normal serum protein, essentially, 7.3 and 7 on two different occasions with an albumin of 2.5 and a globulin of 4.8. The serum bilirubin is interesting. He came in with a total bilirubin of 4 mgs.%, and a direct reading of 2.7 mgs.%, giving an indirect reading of 1.3 mgs.%, and that was almost the proportion of the prompt and indirect reading of the van denBergh test on all three occasions. Later it was 15.4 mgs.% direct and 7 mgs.% indirect which would mean he had a moderate degree of jaundice which deepened to a higher degree. Would this help us any? I believe not because in any type of jaundice, either in obstructive or hepatogenous, you may get that sort of reading. I should say it was entirely nonspecific and merely indicates the presence of hyperbilirubinemia and nothing else. The stool findings are misleading because one stool examined early showed a positive urobilinogen and one taken later showed a negative urobilinogen. They were examined weeks apart. There is no comment made as to the color or these stools, and there is no mention of the urinary urobilinogen. Many cultures were taken and they were all negative. The serum amylase was 19 units; later the NPN was 42 mgm.%, and the Wassermann was negative. The urine was essentially normal except for a trace of albumin and a 1+ sugar. The sugar is interesting because nowhere else in the protocol do we have the mention of a repeat urinalysis, and we have no blood sugar determination to determine whether the urinary sugar is significant or not. The miscroscopic study of the urine was normal and there were no amœbae in the stool.

From the standpoint of his laboratory findings, it seems to me that we can conclude two things only; namely, that he had a low grade leukocytosis and evidence of some pathological state that caused a reversal of the A-G ratio. This, as you know, can be due to any type of disease involving the reticuloendothelial system. It is notably true in liver disease that anything that will raise the globulin may reverse this ratio. The albumin was 2.5 gms.. I do not think anyone knows why there is a reversal of this in hepatic disease. There are many interesting theories, but we just do not know the reason. These are about the only two positive findings that are worth much to me. You might say that the amylase helps us in the determination of pancreatic function and that his urinary tract was essentially normal, but other than that, we cannot definitely say that this represents a significant hepatic disease which is diffuse or intrinsic, nor can we say that it represents disease of the extra biliary tract. That is all I can gather from the facts. To summarize, here is a man taken sick two months before admission with an illness which is characterized by spiking fever, deep jaundiced and hepatomegaly who died within 3 or 4 months, probably in cholemia, and who was found to have on pathologic examination, a nodular liver, and one which under the microscope appeared to show inflammatory reaction within the liver parenchyma and a reaction to that, namely, fibrosis.

Now, the only thing for us to think about is, is the pathologist correct? Does this represent a form of hepatitis with a fibroblastic reaction or does it not? This patient had drunk alcohol moderately over a long period of time; alcohol may be a factor with other things in producing fatty deposition in the liver. He had a nodular liver which is acceptable for a person who has had chronic liver disease with fibrosis of any type. He had a reversal of his A-G ratio, which is characteristic of intrinsic liver disease; he had microscopic changes in the liver cells which were thought to be due to hepatitis.

This may indicate that we are dealing with a patient with hepatitis. Hepatitis, however, is unusual in a person over 60 years of age who has not had some diffuse and definite hepatotoxic agent. You might say that the patient had a chronic hepatotoxic agent. He may have been a cleaner and inhaling carbon tetrachloride over a long period of time or he may have been doing other things, but he was not sick until 2 months before his admission when he became deeply jaundiced. Most patients with hepatitis do not do that unless the illness is acute to start with. This man gives no such history. The length of time he lived (2 to 4 months) is a short time for a person who has that type of hepatitis. If he did have hepatitis, he should have chronic fibrosis of his liver, and if he had chronic fibrosis of his liver, he should have had many stigmata of chronic hepatic disease. He should have had definite varices perhaps and a large spleen, gynecomastia and "liver

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palms" and many other things which this patient did not have. I think that we can say that he has shown none of the evidences of a chronic, long-standing hepatic disease.

Another thing I think important is the type of fever he had; a more or less septic type of temperature. I am personally not familiar with any type of hepatitis in which a septic temperature occurs, with the possible exception of amebic hepatitis. An increased number of circulating leukocytes indicates that you are not dealing with what we commonly term a hepatitis, unless the hepatitis is due to an obstruction in the common duct such as is seen in suppurative cholangitis in which there is an ascending infection into the hepatic ducts, so it would be most unusual, it seems to me, for this man to have had an extensive fibrosis and not have a positive cephalin flocculation. All of these things are definitely against hepatitis.

It may be worthwhile for us to stop a minute now and consider the types of hepatic injury necessary to produce certain pathological changes. For instance, what type of hepatic injury will produce a markedly nodular liver surrounded by fibrosis. It is my understanding that only massive necrosis will produce such a change. This means necrosis of a large portion of the liver involving many lobules in which the reticulum structure is destroyed, and in the effort at regeneration, there is fibrosis also. So it seems to me we have to postulate massive necrosis if we accept this as meaning chronic hepatitis. The ordinary person who has simple viral hepatitis, or the chemical hepatitis that we see, normally has a zonal necrosis. It is true that long-standing irritants, for example, the iron of hemochromatosis, may produce a similar picture. Fatty degeneration over a long period may at many times produce a marked change and a great deal of lobulation. Gummas of the liver may distort the liver tremendously. So there are all forms of hepatitis in which a given injury can produce such a clinical picture as we see here. The thing which concerns me is that we have no indication as to what the etiology may be, where the trauma came from.

Biliary cirrhosis can produce a somewhat similar picture. Biliary cirrhosis, as I have indicated, is always due to obstruction of the common duct or to the outflow of bile into the duodenum, or essentially due to that. I do not think we have any evidence that that is true in this case. His common duct was not obstructed, nor was it even dilated such as it may have been if a stone had been present obstructing the duct and the stone later dropping out. Second, the color of the stools were essentially normal. On one occasion he had urobilin in his feces. However, he had a septic temperature, a leukocytosis, and enlarged liver, no enlargement of the spleen; all are factors that could have produced a biliary cirrhosis of the type which may have produced this picture.

I think we have to consider some inflammatory condition. Tuberculosis produces tuberculomas. He had no evidence of any systemic tuberculosis of any kind. He had leukocytosis and a spiking fever, and this it seems, would be unusual to me with no active focus. We do not have a chest film, which would be of some help. Actinomycosis of the liver has been reported. It is unusual and it is seldom a primary disease in the liver, and the fact that we have an exploratory laporatomy showing that there is no other organ in the peritoneal cavity involved, makes actinomycosis unlikely.

The lymphomas, such as Hodgkin's disease, can certainly produce nodules with perhaps fibrosis around them. However, there was no report made of any enlarged lymph nodes being found in the peritoneal cavity. It would certainly be unusual if his pathology was solely limited to the liver and no other organ. And, in the absence of a chest plate, we have to say that there is no other organ involved that we know of.

Hemochromatosis has to be considered because it does produce irritation and fibrosis as a result of the hemofuscin and hemosiderin deposited within the hepatic parenchyma. This man has a large liver. Usually hemochromatosis is most unusual over the age of 60. Again, it is usually a liver that is smooth, enlarged and granular, but not nodular to this degree. The patient had glycosuria. One begins to wonder whether that is significant or not. We do not know. However, one does not have the other evidence of hemochromatosis so I think we can pretty well discard it as being a possibility.

The protozoa, namely, amoeba, must be considered. This man had no diarrhea; but that doesn't mean much. A great many people who have amoebic abscesses or amoebic hepatitis, do not have diarrhea. Multiple abscesses might produce nodules in the

liver. A single abscess is much more common than multiple ones. It is interesting that in amoebic hepatitis and abscesses, there is always leukocytosis in the early stages. However, it does not seem to me to have the other evidences that we need to consider in a diagnosis of amoebic infection.

Sarcoidosis has to be though of—it can produce any type of granulation in the liver, but again, it would have to be the only organ involved. However, he does have a hyperglobulemia, which is commonly associated with sarcoid disease; however we would expect his spleen to be large and infiltration into some other organs. I shall discard this possibility.

Gummas are to be considered. Dr. Kinloch Nelson made a statement in a meeting I attended a couple of months ago that there was only one case in Dr. Frank Apperly's collection of gummas of the liver. If it is that rare, I think we had just better discard it. It certainly is a rare disease. The patient's Wassermann is negative which doesn't exclude the diagnosis, but even if the Wassermann was positive, it would not be a valid assumption to assume that the hepatic pathology was due to syphilis. It has to be thought about, but it does not seem to me to explain the picture.

I do not believe he had a benign tumor. I think all of us can agree to that. One thinks also of the sarcomas. Sarcomas will act very queerly in a liver, they do not usually produce that type of nodularity. I do not think we have any evidence for this since one would gather that the pathologist would not have much difficulty in differentiating such a condition; I think we can probably rule it out. One should consider metastatic carcinoma. He has nodules in his liver with mild fibrosis, and he dies in deep jaundice with a septic temperature. All of us know that metastatic carcinoma of the liver can mimic anything in the abdominal cavity. However, if this is the diagnosis, where did it originate? His stomach is normal; his colon, I suspect, is normal, since he has no evidence of obstruction or blood loss or anything else. He has no evidence of any other type of primary disease within his peritoneal cavity nor within his lung—I think you have to consider the lung carefully; all of us have seen a small primary pulmonary lesion in the bronchus, manifest itself only in the peritoneal cavity in the liver therefore, you have to consider something like an oat-shaped cell tumor which is very difficult at times to differentiate, and which one must always call to mind and think about. We have no evidence to indicate that the patient might have a primary tumor in the lung.

The pancreas must also be considered. He had a type of pain which could be due to pancreatic disease. Usually, if you have a pancreatic tumor that is causing jaundice, you have a typically obstructive jaundice. It would be difficult, therefore, to say that this man had a cancer at the head of the pancreas with the type of jaundice he manifested. Had he had a carcinoma in the tail of his pancreas, he would not have had any jaundice at all, except from possible metastasis from the tail, and it seems to me that that could happen, but by that time, I think he would also get continuity of the tumor into the head and probably would have obstructive jaundice, which this man did not have. The loop of the duodenum was not enlarged. He did have a little sugar in his urine on one occasion and it is interesting to note that people with carcinoma of the pancreas will run sugar on one test and none on the next so at least this diagnosis has to be entertained as a possibility. The other consideration is a primary carcinoma of the liver, and here we are dealing with a difficult diagnosis at any time. This man we will say had a high degree of icterus—I think we can say that in primary cancer of the liver, one of the common things is that a patient gets along pretty well, and suddenly begins to go downhill with icterus becoming more pronounced, and usually there is evidence of increased portal tension, etc. This patient had a deep jaundice which continued, he had weight loss, he had pain which would be acceptable, and he had moderate fibrosis in the liver. We all know that fibrosis is a common accompaniment of primary cancer of the liver, but the amount of fibrosis is important. This man, if he had had a typical cirrhosis for a long period of time, would, I believe, have shown many more changes in his liver, than were reported by the pathologist on the biopsy. Hepatoma more often occurs in people who have diffuse, intrinsic liver disease for a long period of time. It is common to have fever; it is common to have leukocytosis, and I do not think from the evidence we have that we can discard it. Against it, however, is the fact that we have a pathological specimen which shows hepatitis plus fibrosis. Well, this is very interesting, but I have been told many times by a pathologist that the cells of hepatoma are

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identical to the normal polygonal cells of the liver so that at times, the hepatoma just resembles a normal liver so to speak. Thus, it is possible that the pathologist could have been looking at a nodule of polygonal cells, which look like normal ones. They were cells of a new tumor or neoplasm or a new liver tying to be formed by the hyperplastic reaction from neoplastic cells. One of the common ways a hepatoma manifests itself is that it is similar to a liver abscess in that it produces fever leukocytosis. Therefore, despite the pathological report on the biopsy, I believe that the information I have fits the picture of a primary hepatoma grafted on a liver which has had some previous hepatic pathology, but not major pathology. I would favor hepatoma over cholangioma only by the fact that there is more fibrosis with the former, as a rule, because it is more commonly associated with the cirrhotic syndrome. However, I do not think we can say which it was. I do not think we can entirely throw out biliary cirrhosis. It worries me a little about the cerebral change, with his pupillary abnormalities, whether or not he may have something in his cerebrum to account for it, I do not know.

HOSPITAL DIAGNOSIS AT TIME OF DEATH: (1) Subacute yellow atrophy (2) cirrhosis.

DR. CARAVATI'S DIAGNOSIS: Primary carcinoma of the liver.

PATHOLOGICAL REPORT: DR. GORDON R. HENNIGAR*.

The liver, at autopsy, weighed 2,000 grams, but appeared to be somewhat smaller than normal. On close inspection, the organ was green in color and consisted of prominent lobules separated by quite apparent translucent connective tissue. Undoubtedly, the latter contributed greatly to the weight of the organ. On close inspection the whole liver was the "seat" of fine nodularity (Fig. 1). Occupying the hilar region was a poorly defined, firm, white mass measuring 5 to 7 centimeters in widest diameter. At necropsy, it was felt from gross observance, that the tumor would be arising from a large, intrahepatic bile duct. The gallbladder and extra-hepatic ducts showed no obstruction.

Microscopically, one sees the appearance presented by a longstanding obstruction to the outflow of bile. The intralobular canaliculi are dilated and

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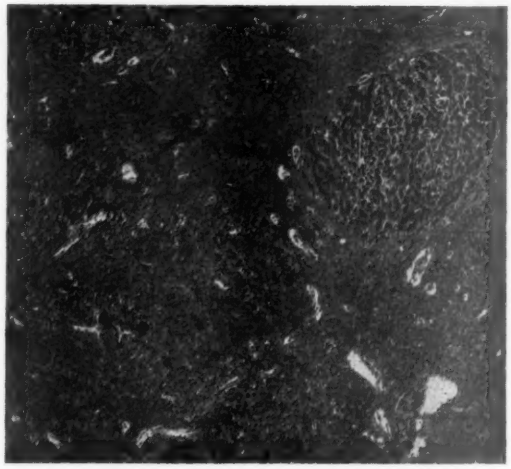

Fig. 1. Note isolated lobules separated by connective tissue.

contain "bile thrombi" or "bile cases". Frequently, these are sizeable (Fig. 2). Many of the adjacent liver cells are small, contain cytoplasmic granules and considerable bile pigment. Sudanophilic material (lipid) is seen in some cells, but is far from abundant. The amount of reticulum and collagen of the supportive framework of the lobule and portal areas is ostensibly in excess of normal. In the portal areas one is struck by great numbers of bile ducts and ductules (Fig. 1). These were most probably derived from pre-existing bile channels by hyperplasia (proliferation), however, their origin from lobular hepatic cells is always an entertaining idea. No

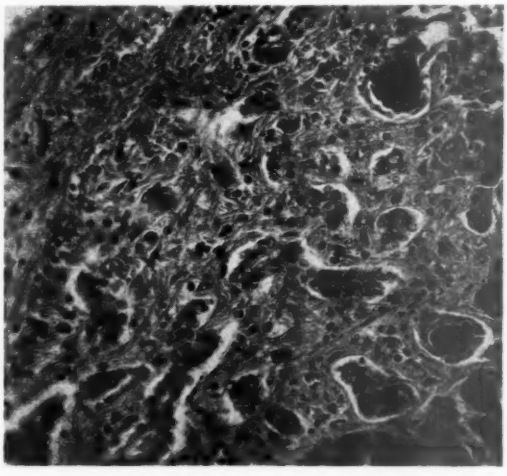

Fig. 2. Proliferative bile ductules and "bile thrombi" are evident.

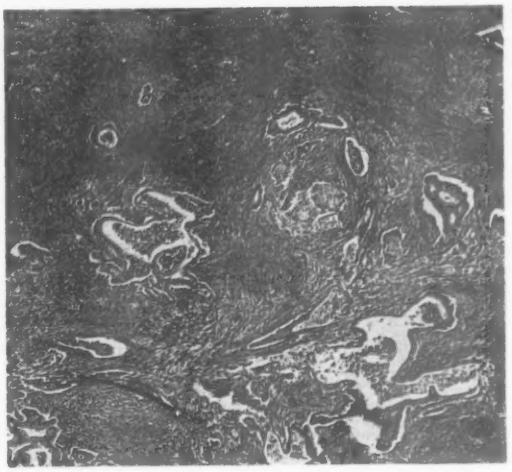

Fig. 3. Malignant glandular structures ramifying throughout, amongst the scarred areas.

mitoses were seen in these proliferating cells.

Sections of the neoplasm reveal it to be composed of malignant cells arranged in a glandular pattern, surrounding spaces, a few of which contain bile (Fig. 3). The cytoplasm is plentiful and the cells are arranged in an orderly fashion. The nuclei show a moderate degree of variation in size, shape and staining qualities. Prominent nucleoli and atypical mitotic figures are not difficult to find. Extremely bizarre mitotic figures and large tumor giant cells, so frequently found in hepatocarcinoma (hepatoma), are conspicuous by their absence. A feeling of inadequacy, when dealing with these tumors, often descends upon we morphologists. Not infrequently, we see histological patterns which in one field will resemble a bile duct adenocarcinoma (cholongioma) and in another be reminiscent of hepatocarcinoma (hepatoma). Of course, it is possible to have two different carcinomas in the same liver. Lastly, we all are familiar with the more than coincidental occurence of Laennec's cirrhosis (portal cirrhosis) and hepatocarcinoma. It has been estimated variously in different series that hepatocarcinoma is superseded by portal cirrhosis in 65 per cent to 85 per cent of cases. Very little is ever said concerning the finding of hepatocarcinoma in a liver, the "seat" of biliary cirrhosis. Recently, I had the opportunity of studying two such cases.

Certainly, there is much left to be desired in our understanding of the role and relationship of biliary cirrhosis, portal cirrhosis, and post-infectious hepatitis cirrhosis, to the development of the carcinoma of the liver.

PATHOLOGICAL DIAGNOSES:

1. Primary intrahepatic adenocarcinoma bile ducts (cholangioma).
2. Biliary cirrhosis.
3. Bile nephrosis.
4. Bronchopneumonia.
5. Testicular atrophy.

Floral Eponym

Forestiera

CHARLES LE-FORESTIER

Forestiera is a genus of 20 species of New World shrubs of the olive family. F. acuminata goes by the common name of swamp privet.

Charles Le-Forestier was a French physician and naturalist who flourished about 1800.

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MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals.

Care of Syphilitic Patients at Central State Hospital*

The incidence of syphilis in the new admissions to this hospital is high, therefore, there is an adequate opportunity to study and compare the various types of treatment for this condition.

During the first week following admission to the hospital, complete physical and laboratory examinations are done on each patient. Of the 1,003 new admissions for the year ending June 30, 1950, 291 or 29% were found to be infected with syphilis in some form. Of these 106 or 10.5% had syphilis of the central nervous system and 185 or 18.4% syphilis in other forms. Though this percentage appears large it is a decided decrease from previous years and is probably due to more effective treatment at the State Health Centers.

Treatment here is promptly started as soon as the diagnosis is made and confirmed. For those who have syphilis other than of the central nervous system, our treatment consists of 7,500,000 units of penicillin given 300,000 units for twenty-five consecutive days and at the same time mapharsen .06 grams each week for twelve consecutive weeks, followed by bismuth 1cc for twelve consecutive weeks. This is followed by a rest period of two months at the end of which time a check is made of the blood Kahn and Kolmer and if found still positive, another course of mapharsen and bismuth is given. If negative they are rechecked in three months and then at six month intervals.

When the psychotic condition of these patients improves sufficiently an effort is made to get them home and a copy of our laboratory findings and treatment record, with a request for follow-up checks and treatments, is sent to the Health Department in the city or county where the patient lives.

Those found to have central nervous system syphilis are given 7,500,000 units of penicillin in conjunction with fever cabinet treatments, 10 to 15 treatments being given at a temperature of 105 degrees over a period of six hours at weekly intervals. While under cabinet treatment mapharsen is given while in the cabinet and after completion of the cabinet treatments the patient is started on bismuth. When this is completed a recheck examination of the blood and spinal fluid is made. When a patient has shown sufficient improvement mentally, he is permitted to leave the hospital at the completion of the above outlined procedures, and a copy of our laboratory findings and treatment and the name of patient is sent to the Health Department in his home county or city with a request for follow-up checks and further treatment if necessary.

Some of our patients who are found to have central nervous system syphilis are inoculated with malaria, the quartan type being used. In these cases we usually proceed with the penicillin as outlined above, this to be followed with malaria inoculation and then mapharsen and bismuth is used following the completion of the malaria therapy.

In some cases we have felt that malaria therapy was superior to the artificial hyperpyrexia but the latter is easier to control if the patient doesn't react well.

Of the 106 patients who had syphilis of the central nervous system during the past year, 67 recovered or improved sufficiently to be released from the hospital with 14 returning to the hospital for further treatment. However, after remaining a while and receiving additional treatment, a number of those were able to leave again.

When our patients are admitted early in the course of the disease so that we can start treatment before too much brain destruction has taken place, nearly all improve to such an extent that they can return to their homes, and, in many cases, resume their former occupations.

We feel that the results obtained here by the above outlined treatments will compare favorably with those done anywhere in the country.

We have been particularly careful to have our laboratory procedures here checked. The blood and spinal fluid in each case is examined both here and in the State Laboratory in Richmond and when there is a discrepancy between the reports we always have a re-examination made in both laboratories.

*Article prepared by M. S. Brent, M.D., Superintendent of Central State Hospital, Petersburg, Virginia.

PUBLIC HEALTH

L. J. ROPER, M.D.

State Health Commissioner of Virginia.

The Physician and Morbidity Reporting

Since the practicing physician is the source of morbidity reports, it is obvious that an effective reporting system implies on his part a realization of the importance of prompt reporting and harmonious working relationships between him and the local and State health departments.

Accurate reporting of communicable disease morbidity is essential for epidemiological investigation and analysis of:

(1) Communicable disease trends

(2) Sex-race distribution

(3) Geographical and seasonal occurrence

Uniformity in reporting is necessary in order that valid comparisons of disease in one locality or state can be made with other states, with the country as a whole and with other countries. This is particularly true in our modern world where, with rapid transportation facilities, persons who have been exposed and contracted communicable disease in one continent can easily reach another continent in the first days of the incubation period.

Our modern system of reporting is only three-quarters of a century old. In 1870 local boards of health began to require reporting of the more important contagious diseases. Tuberculosis had long been considered a family taint, but gradually through public education it began to be thought of as a communicable disease and was made reportable in the mid-nineties.

Earlier attempts at communicable disease reporting had been incidental. As early as 1741, Rhode Island required tavern proprietors to report contagious diseases among their guests and in 1821 Louisiana State Law required similar reporting from keepers of taverns and boarding houses.

Prompt and reliable reporting assumes a new and vital importance in a period when we must consider and prepare for defense against possible biological warfare. Such warfare by sabotage of water, of food supplies or by aerial contamination of buildings could produce serious consequences. Inadequate as our present defenses may be, the importance of epidemiological intelligence and the investigation of all epidemics as they occur is obvious. Only with prompt information can immediate control and defense measures be undertaken.

Morbidity statistics are our most delicate measure of the control of communicable disease. An adequate reporting system must be prompt and dependable—one that utilizes to the full the facts concerning disease incidence currently known to physicians, hospitals, and laboratories. In a world as closely knit as ours is today, time is of the essence.

THE MORBIDITY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	April 1951	April 1950	Jan.-April 1951	Jan.-April 1950
Brucellosis	4	10	20	23
Diarrhea and Dysentery	119	222	748	1,174
Diphtheria	8	11	52	79
Measles	3,075	499	7,154	1,012
Meningitis (epidemic)	11	14	55	61
Poliomyelitis	1	3	14	13
Rocky Mountain spotted fever	0	0	0	0
Scarlet fever	99	84	578	420
Tularemia	1	2	18	18
Typhoid and paratyphoid	2	4	18	24

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MEDICAL SOCIETY OF VIRGINIA

MINUTES OF THE COUNCIL

The Council of the Medical Society of Virginia was called to order on April 26, 1951, at 1105 West Franklin Street, Richmond, Virginia, by the President, Dr. C. L. Harrell. Dr. Walter A. Porter gave the invocation. Dr. Harrell then gave his report to Council in which he welcomed them to the new house and expressed gratitude to the Richmond Academy of Medicine for their aid in the past. Dr. Harrell gave a resumé of his activities during the past months, as representing the Society in functions all over the State.

The roll was called; those present were: Dr. Harrell, Dr. John T. T. Hundley, Dr. M. Pierce Rucker, Dr. L. J. Roper, Dr. Walter P. Adams, Dr. Guy W. Horsley, Dr. Wilkins J. Ozlin, Dr. Walter A. Porter, Dr. Frank A. Farmer, Dr. Vincent W. Archer and Dr. James P. King. The minutes of the Council meeting, January 18, 1951, were then read and approved.

Dr. Harrell told of his conversation with Dr. Kinloch Nelson, with reference to the meeting scheduled by the Department of Clinical and Medical Education by the Mid-Tidewater and the Northern Neck Medical Societies at Tappahannock recently. This was elaborated upon by Dr. Malcolm H. Harris.

Dr. Adams told of progress in plans for our convention at the Cavalier Hotel in October, and there was considerable discussion of a number of detail items, such as scarcity of rooms, schedule of meetings, arrangements for the banquet, speakers, etc. It was found to be the sense of the Council that one-half hour period should be set aside for a presentation of the American Medical Association and its activities to be followed by discussion.

The Council was told of opposition to beginning the meeting on Sunday, but this was tabled for consideration by the succeeding President. Dr. King offered a resolution proposing the development and distribution of a Directory of officers, committee members, and possibly the entire membership of the Medical Society of Virginia, the cost to be considered. A committee of Dr. James P. King, Dr. Vincent W. Archer and Dr. Frank A. Farmer was named to study this.

The Council gave considerable thought to the time of meetings, the election of officers, and the report of membership by local medical societies, with the view that some benefit might accrue from more uniformity in this regard so far as furnishing the State Office with pertinent information in due time. It seems highly desirable that officers for the current year and membership of local medical societies be reported to the State Office by December 1 of each year. The proposal of making an effort to include the membership of the State Society in the Roster of Physicians and Surgeons, recorded each year by the Board of Medical Examiners, was discussed. Someone raised the question if this could not be done in the MONTHLY. Dr. Archer pointed out this had been tried before and indicated that the cost of such a roster in a Directory might be considered.

Dr. Harrell called on Dr. Malcolm H. Harris, Chairman of the Ethics Committee, for a report. Dr. Harris presented a resolution from his Committee as follows: "The Ethics Committee of The Medical Society of Virginia unanimously concurs in the principles of medical ethics covering The Medical Society of Virginia, which are in compliance with those of the American Medical Association. These condemn any manner of so-called 'kick-backs' or 'fee-splitting' to physicians. This ethical principle is as follows: . . . The acceptance of rebates on prescriptions or appliances, or of commissions from attendants who aid in the care of patients is unethical. An ethical physician does not engage in barter or trade in the appliances, devices or remedies prescribed for patients, but limits the sources of his professional income to professional services rendered the patient. He should receive his remuneration for professional services rendered only in the amount of his fee specifically announced to his patient at the time the service is rendered or in the form of a subsequent statement and he should not accept additional compensation secretly or openly, directly or indirectly, from any other source'."

Dr. Harris indicated that judging from reports of Richmond newspapers, there seemed to be some feeling that this was a matter that the General Assembly of Virginia should handle. Dr. Harris expressed the Committee's view that this was a matter that should be handled within the profession and his letter to the Governor on this subject was read. The Governor's reply indicated his confidence that the profession could handle it.

Dr. Harrell referred to the newspaper publicity in Norfolk on this and told of his reply to it.

Dr. Harrell mentoned this practice prior to 1916 in Norfolk and the action of the Norfolk County Medical Society, then. Dr. Horsley referred to progress made in the Richmond area since 1945.

Dr. Adams suggested the advisability of publicity on "Medical Society of Virginia denies 'kick-backs' in the last five years."

Dr. Harris thought of the importance of approaching this through the component societies, though it was stated that some of them did not have this to deal with. It was also brought out that if the local society did not handle it, the State Society should.

Dr. Archer inquired of the wisdom of expulsion from the society of any who persisted. Dr. Farmer felt that some burden of proof was on those who had been accused.

It was deemed advisable to write those whose names

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appeared in the court record indicating that the Council desired to clear this matter. It was agreed that the office should do this with the approval of the Ethics Committee and that a statement should go to the Society of Oto-Laryngology and Ophthalmology. Dr. Hundley offered a motion that a copy of the report of the Ethics Committee and a statement of its approval by the Council be sent to the Secretary of each component Society and to certain special societies and that they be requested to read these to their membership requesting that evidence of a continuation of this practice be sent to the Ethics Committee. This was passed. Dr. Archer's motion requesting the Oto-Laryngology and Ophthalmology Society to condemn such practices was passed. It was agreed that a statement on this subject should be prepared for the press.

Dr. Harrell then asked Dr. King to discuss the American Medical Education Foundation. Dr. King referred to the beginning of this movement in Cleveland and the appropriation to this by the Medical Society, and said after some discussion of this, he and Dr. Harrell felt that it was a matter that should come before the Council. In the meantime, he had discussed it with the deans of our two medical schools. Both deans preferred that funds come directly from within the state, but thought some Federal assistance would be necessary. Dr. Harrell referred to the results of a poll of the Council. Dr. Archer referred to correspondence with Dr. Elmer Henderson, in which it was stated that funds earmarked for medical schools of one's choice would be turned over directly to them. Dr. Adams pointed to the merit of this being handled by individual societies. Dr. Archer inquired of the wisdom of contacting each member through their Alumni Association, encouraging them to subscribe to the school of their choice. He also said that Dr. Rucker suggested that Dr. King write an editorial for the Monthly. Dr. Harrell thought it should be left up to the local component societies. Dr. Hundley felt that it should have the approval of the State Society. Dr. Adams felt that Dr. King's editorial could take care of this.

Dr. Harrell requested Dr. Rucker to report for the Program Committee. Provision had been made for approximately 40 papers and guest speakers. There was some discussion of the printed program used by the General Practitioners. Dr. Rucker indicated that they wanted to designate the time for the papers this year, so that physicians could plan their attendance, and emphasize the importance of having the program run on schedule. Dr. Harrell mentioned that colored physicians can attend meetings in the Panorama Room.

The role of non-members of the Medical Society of Virginia holding office in local medical societies was discussed, but it was agreed that local autonomy should prevail.

Considerable discussion followed on the subject of inactive societies, but no action was taken. Dr. Harrell suggested that the Secretary try to revive interest in these societies.

Dr. Harrell then brought up the subject of stimulation of work of the various committees of the society. After considerable discussion, Dr. Hundley thought that work should never be relegated to a special committee on a subject in the field covered by a Standing Committee. Dr. King disagreed. He felt that things to be done should be given to interested people.

Dr. Ennion S. Williams also reported on some progress of the program committee. He indicated that tentatively it had been decided to postpone planned discussions after general sessions until after the sectional meetings. He gave a tentative outline of the program. There was some discussion of their efforts to arrange the schedule. It seemed that the proposed medical Examiners Panel might not be arranged before the last day. Dr. Hundley liked the idea of a post-convention Council meeting. Dr. Archer inquired of the date of specialty luncheons. Dr. Rucker indicated a time had been recommended. Dr. Harrell expressed a desire that the program Committee would use Vice-Presidents in presiding.

Dr. Archer, Dr. Porter and Dr. Harrell inquired of our expenditures and income.

A financial report showing receipts of $61,923.90 and disbursements of $79,785.26, which included $27,371.06 spent for and on the house to date.

The question of annual dues paid by Health Officers arose, this was construed to constitute a hardship in some instances, though no arrangement was agreed upon.

The subject of converting to oil heat in the house was brought up and Dr. Archer inquired of the operating expenses. Bids on converting to oil heat were read. Dr. Roper felt that comparable items should be covered in competing bids. Dr. Archer offered a motion that Council authorize the House Committee to proceed with installation of oil burning equipment, not to exceed $2,500 with outside oil storage. Dr. Roper was added to the House Committee. Dr. Harrell inquired if we should rent any of our space.

The question arose about the need of space on the second floor for the Medical Society of Virginia and possible rental of any unused space. The House Committee was empowered to rent out any space not needed above the first floor.

A motion was passed that we proceed with an application for exemption from taxes.

The question of insurance on the House was discussed and the Council agreed that the insurance should be increased to 75 per cent of the estimated value.

It was explained that our janitor had left us. This matter was left to the House Committee.

Dr. Porter inquired about any thought of re-districting, but it was agreed that nothing could be done until the legislature meets.

Dr. Harrell raised the question of an advertiser furnishing the program. A motion was passed that this be of the book-type, similar to that used by the General Practitioners provided the program committee wants it. It was pointed out that if this were done, all advertisers

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should be advised of it.

Dr. Warthen then appeared to discuss any matters for the house committee. Some discussion ensued with respect to any changes to be made on the second floor and the committee was authorized to proceed with improvement in the basement, not to exceed $750, after the installation of the oil burner.

It was agreed that an issue of the MONTHLY feature the House or office and the Secretary was instructed to prepare some material on this.

Dr. King brought up the question of titles for office personnel. Dr. Archer moved that Miss Watkins be given the title of Executive Assistant. This was passed. There was some discussion of a title for Mr. R. H. Nash, but it was felt that the Public Relations Committee and the Secretary should be consulted on this matter.

Since there was no further business, the meeting then adjourned.

HENRY S. JOHNSON,
Executive Secretary-Treasurer.

A more comprehensive coverage of the detailed minutes is on record in the office.

PUBLIC RELATIONS

Council Convenes

Under the chairmanship of President C. Lydon Harrell, the Council of The Medical Society of Virginia convened April 26 at the new state headquarters at 1105 West Franklin Street, Richmond. Many expressed satisfaction with the adequacy, comfort, and attractiveness of the office.

Following Dr. Walter Porter's invocation, Dr. Walter Adams announced progress in making local arrangements for the forthcoming Society convention at the Cavalier Hotel. Lack of adequate space will reduce this year's income from technical (commercial) exhibits.

Programming reports made by Dr. M. Pierce Rucker and Dr. Ennion S. Williams indicate that some forty papers are expected to be presented. Plans are being made to announce the agenda in such a manner as to enable members to attend the sessions of most interest to them.

Dr. James P. King was selected to head a committee to study the feasibility of publishing a handbook containing the officers, committee chairmen, roster of members and other pertinent Society information.

Dr. M. H. Harris, Chairman, Ethics Committee, reported the results of the Committee's consideration of current references to kickbacks. The Council approved what had been done by Dr. Harris' group and directed that it continue its fact finding work regarding kickbacks, reporting to Council at the next meeting.

Various matters concerning the new office building were presented by Dr. Harry J. Warthen, Jr., Chairman of the House Committee. Authority was given to install a new heating plant and remodel certain basement space to afford adequate storage facilities.

At least a dozen other important items were discussed by the Council in a busy day's session.

Comprehensive, detailed minutes are kept in the office and a condensation of these are carried elsewhere in this issue of the MONTHLY.

Society Structure

Chosen annually by the component societies, the House of Delegates is the policy making body of The Medical Society of Virginia.

While not in session, the House of Delegates affairs are conducted by the Council in an executive-administrative capacity.

Normally convening three times yearly, nine Councilors—one from each Congressional District—in addition to the Society President, President-Elect, First Vice-President, the Immediate Past President, the Editor, and the State Health Commissioner compose the Council. Incidentally, if Congressional District lines are to be followed, it may be that some thought should be given to provision for the tenth member of Council in 1952.

In emergencies or in instances not of sufficient import to convene the entire body, an executive committee of the Council advises with the President. This committee consists of 3 members appointed annually by the President.

To comply with the State Society By-Laws, Members of the District Council should be elected not later than September 30 of each year. In instances

where this is not done, the Councilor should appoint these.

Headed by the respective Councilors, each District has a District Council. Seemingly, some degree of confusion exists as to the distinction between members of the District Council and Councilors. Following the provisions of the Constitution and By-Laws, there is a Member of the District Council representing each component medical society and one for each county in which there are physicians but no medical society. These work with and under the direction of the Councilor of the respective districts. Members of the District Council perform an important function in linking the membership with the state Council.

Holding annual conferences for the past few years, and participating in the annual Public Relations conference, these Members of the District Council have been in a better position to be informed and to assist the membership of local medical societies in matters of current interest coming to the attention of the state Society.

Physician Placement Program

"The Alumni Association was called upon by the citizens of Coalgate, Oklahoma, to aid Coalgate and vicinity in securing the services of a physician. The Alumni Association located a fine young physician who was interested in establishing his practice in Coalgate. However, the young physician, Dr. Wallace Byrd, could not go into general practice without some financial aid. The Alumni Association provided the aid. Below is reproduced a letter which the Alumni Association has recently received from Dr. Byrd, at the end of his first year of practice in Coalgate:

" 'It is a pleasure to be able to repay the University of Oklahoma School of Medicine Alumni Association the amount so generously advanced me to assist me in establishing myself in practice at Coalgate, and to be able to report to you that your forecasts of what could be done in this location have proved well founded. My practice is continuing to grow and I am presently approaching the point where I can see my office comfortably well equipped for the practice of internal medicine as a part of my general practice.'

"It is the feeling of the Alumni Association's Officers and Trustees that no finer gesture by Medicine in Oklahoma could do more to thwart the inroads of Socialized Medicine than by continuing activities of this nature."

The above article was sent the Society office by Dr. Andrew F. Giesen, member of the Public Relations Committee of The Medical Society of Virginia.

Temporary Physician Services.

Each year several requests come to us from physicians, licensed to practice in Virginia, for help in locating from one to three months during the summer.

An effort is being made to be of assistance to both those wishing to locate here and to those desiring the temporary services of physicians.

Cooperation by members is solicited.

Dues Exemption.

In accordance with what appears to be a national trend among medical associations, The Medical Society of Virginia Council has directed that payment of dues by members temporarily serving in the armed forces not be requested.

Venereal Disease Control Committee

Interpretation of the Blood Serologic Tests for Syphilis in Diagnosis and Treatment

Facts you should know about these tests in diagnosis:

1. About 1 in 4000 of the normal population shows a false positive blood test.

   (Note: the incidence in hospital populations is somewhat higher)

2. Some diseases other than syphilis are regularly associated with positive tests. (yaws, pinta bejel)

3. Some diseases are frequently associated with positive tests (leprosy, malaria, infectious mononucleosis, post-vaccination state, virus pneumonia).

4. Some diseases are occasionally associated with positive tests (trypanosomiasis, rat bite fever, leishmaniasis, kala-azar, disseminated lupus erythematosus, genital herpes, lymphogranuloma venereum, chancroid, chicken pox, subacute bacterial endocardi-

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tis, infectious hepatitis, febrile states of varied etiology).

5. Some diseases may questionably be associated with positive tests (tuberculous toxemia, scarlet fever, measles).

6. Positive tests may occur in hyperglobulinemia, following foreign protein injection, in blood donors after several donations.

This should not discredit these tests since they are surprisingly specific and accurate if properly interpreted. Fortunately the false positive tests are either low titred or transient in nature (3 months or less).

Suspect in diagnosis a false positive test:

1. If there is disagreement between the precipitation test and the complement fixation test.

2. If the readings are low titred (to the quantitative procedure which is absolutely essential for proper serologic diagnosis).

3. If on repetition of the test within a few weeks there is obvious change in titre downward without treatment.

4. If there has been any possible clinical reason for a false positive test (See above 1-6 on the basis of the history or physical examination.

One may generalize for practical clinical diagnostic purposes that a repeatedly high titred quantitative reading means syphilis in the absence of evidence to disprove, whereas a low titred reading (8 units or less) means probably no syphilis particularly if there is fluctuation of readings and on repetition the reaction becomes less positive. (Note: There are occasional exceptions to this statement.)

It cannot be sufficiently stressed that it is better to repeat inconclusive tests over a period of months if necessary rather than make an erroneous diagnosis since treatment is usually not of an emergency nature if the person is asymptomatic (except syphilis in pregnancy). A spinal fluid examination may be necessary as part of the diagnostic work-up.

At times even under the best of circumstances results may be inconclusive. However, certain trends in diagnosis are reducing still further this inconclusive percentage; e.g., Neurath euglobulin inhibition tests, treponema immobilization test and the use of cardiolipin antigen. The first two procedures are still too experimental and difficult for widespread adoption; the third has been widely adopted.

One must also realize the possibility of a false technical positive reading (laboratory error, incorrect recording) since these tests are usually done in large numbers at a time.

Facts You Should Know About Interpretation of Serologic Tests After Treatment

1. In general the longer a person has had syphilis the more likely the tests will remain "fixed" or unaltered by treatment e.g.

a. In early syphilis (less than 4 years), the tests in a satisfactory treatment response usually became negative 3 months to one year after treatment has been concluded; therefore,

1. Don't expect any striking change in titre during or immediately after treatment.

2. Take monthly quantitative tests for one year after treatment. This will show the trend of the titre.

3. Continue serologic follow-up after the first year every 3-6 months for at least five years so that any relapsing case may be discovered by sudden definite rise in titre.

4. Note: There will be a small percentage of successfully treated cases of early syphilis whose titre will stabilize at a low level (4-8) units rather than negative.

b. In late syphilis (four or more years duration) do not expect any appreciable change in titre after treatment. Success of treatment depends upon periodic clinical reevaluation, and in central nervous system syphilis upon spinal fluid findings as well.

The problem of interpretation of spinal fluid findings is beyond the scope of this presentation. Briefly, it should be mentioned that a spinal fluid examination should be performed before treatment in all late cases of syphilis and 3-6 months after treatment in early syphilis.

HARRY PARISER, M.D., *Chairman*

WOMAN'S AUXILIARY TO THE MEDICAL SOCIETY OF VIRGINIA

President ..... MRS. C. M. McCOY, Norfolk
President-Elect .. MRS. HERMAN W. FARBER, Petersburg
Recording Sec'y.. MRS. L. BENJAMIN SHEPPARD, Richmond
Corresponding Sec'y.... MRS. LEMUEL MAYO, Portsmouth
Treasurer..... MRS. KALFORD W. HOWARD, Portsmouth

Northampton-Accomac.

The Auxiliary to the Northampton-Accomac Medical Society held its spring meeting at the home of Mrs. W. L. Cosby, Painter, on April 10th. Guest of honor was Mrs. C. M. McCoy, Norfolk, president of the State Auxiliary.

A delicious dessert and social period preceded the business meeting and each member was presented with a beautiful camelia with the compliments of the president, Mrs. W. T. Green, Jr., these being grown by Dr. Green.

At the business meeting, it was voted to give $10.00 to the Cancer Control Fund and $5.00 for an Auxiliary gift to the new offices of the Medical Society of Virginia in Richmond.

Mrs. McCoy then spoke on "The Art of Being a Good Doctor's Wife".

Mrs. W. C. Henderson, chairman for the Grace Wilkins Memorial Room in the Northampton-Accomac Hospital reported that venetian blinds had been installed and the memorial plaque placed on the door.

The next meeting of the Auxiliary will be with Mrs. C. E. Critcher at New Church on July 10th.

CATHERINE R. TROWER (MRS. HOLLAND)
Chairman, Press and Publicity

Petersburg.

A report on the board meeting of the Auxiliary to the Medical Society of Virginia was given by Mrs. Carney C. Pearce, Jr., at the April meeting of the Auxiliary to the Petersburg Medical Society, held at the home of Mrs. Meade C. Edmunds. Mrs. Palmore Irving and Mrs. W. S. Grizzard were co-hostesses.

Plans for contacting local junior and senior pupils interested in nursing was discussed. The Easter egg hunt, sponsored in March, proved so successful that it may become a yearly project. It was announced that a rummage sale will be held May 10-12 to raise funds for the budget. It was voted to send $20.00 to the Jane Todd Crawford Fund and $10.00 for the Auxiliary Gift to the new offices of the Medical Society of Virginia in Richmond.

A luncheon meeting will be held on May 29th and Mrs. C. M. McCoy, president of the State Auxiliary, will be the guest speaker.

BOOK ANNOUNCEMENTS

Color Atlas of Pathology. Hematopoietic System; Reticulo-Endothelial System; Respiratory Tract; Cardiovascular System; Liver; Alimentary Tract; Kidney and Urinary Tract; Musculoskeletal System. Prepared under the auspices of the U. S. Naval Medical School of the National Naval Medical Center, Bethesda, Maryland. Philadelphia, J. B. Lippincott Company. 1950. xi-546 pages. Illustrated with 1053 figures in color on 365 plates. Cloth. Price $20.00.

This book "Color Atlas of Pathology" fulfills the purpose for which it was intended. Namely it is an atlas of pathology composed of colored gross specimens and microphotographs. The hematology section is composed of excellent pictures of blood smears in the various diseases of the hematopoietic system. The atlas covers a wide scope of diseases and the legends accompanying the pictures are brief, specific, informative and to the point. It is unfortunate that many of the beautiful gross pictures are small and one can make out detail only with strenuous effort. The X-rays of the various lung lesions are particularly clear and well done. The pictures taken clinically to show lesions of the oral cavity and its contents are indeed a work of art. The atlas due to its cost has limited usefulness. However, its excellence can hardly be challenged and it should be high on the list of useful books for supplementary reading for medical students.

G. R. H.

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EDITORIALS

Ennion Gifford Williams (1874-1931)

IF EXCEPTIONAL capacity for work be a sign of genius, the subjects of these sketches were geniuses. All were scientists, true searchers after truth. With the exception of the non-medical discoverers of anesthesia, they seemed not to have been actuated by any money motive. Most of them showed remarkable wisdom in matrimony, marrying women who played an important, although inconspicuous, part in their careers. All had character in a remarkable degree and in most instances, the word "beautiful" could be properly applied to it. Perhaps the one who combined the above qualities in the most harmonious proportions, was the first Health Commissioner of the State of Virginia.

Ennion G. Williams was born in Richmond, January 31, 1874, the son of John L. and Maria Ward (Skelton) Williams. He attended the University of Virginia from 1892-97, graduating in medicine in the latter year. Upon his return to Richmond he became Professor of Pathology, Bacteriology and Histology in the Medical College of Virginia. He also engaged in the private practice of radiology, which was then in its infancy.

Public health work was also in its infancy, there being few specially trained men anywhere in the country. Dr. Williams became interested in the health movement and tried to get the health department of the City of Richmond organized along modern lines. This was a difficult undertaking. In order to put it across he ran for Council. He served on the City Council from 1905 to 1908, and succeeded in electing Dr. E. C. Levy as bacteriologist of the City Board of Health and finally as chief health officer. In the meantime, the State Legislature had created a State Department of Health. Dr. Williams tried to have Dr. Levy appointed commissioner as he was the only available man who had had special training, but for some reason Dr. Levy was not acceptable to the Governor. It looked as though the movement were doomed to failure for the want of a suitable head. In the crisis, pressure was brought to bear upon Dr. Wililams who gave up a lucrative private practice to accept the position at a considerable financial loss. He was appointed Commissioner in 1908, a position he held until he died June 6, 1931.

When Dr. Williams began to build a state health department he had an appropriation of $40,000 per annum. It was quartered, laboratories and all, on the first floor of an old neo-classic house on Capitol Street. There were few precedents to guide him. In an address in 1930 (*Am. J. Public Health* **20**: No. 12, 1930) he described the steps in the development of the Virginia State Health Department. They fell into three categories, education, epidemiology and laboratory. Since the act of the Assembly creating the State Health Department specifically directed that a sanatorium for the study and treatment of tuberculosis be established, and since tuberculosis was the major health problem at the time, the Catawba Sanatorium was one of the first accomplishments. During his lifetime two other sanatoriums for tuberculosis were established, "Blue Ridge Sanitarium," and "Piedmont" for negro patients.

The prevalence of typhoid fever and water borne diseases led to the establishment of the Bureau of Sanitary Engineering. In 1912 a survey, with the aid of the Rockefeller Foundation, showed the need of rural sanitation. It was shown that only 0.3 percent of rural homes had sanitary sewage disposal. Accordingly, a division of rural sanitation was begun, and this in turn led to a co-operative plan of the county, the state

health department and the U. S. Public Health Service for a sanitation officer. Dr. Williams' five point program for school children was known all over the country. In 1918 the General Assembly passed a law that required every school teacher to have passed an examination on physical education and physical inspection of school children. The five points were: eyes, ears, nose, throat and teeth. If these were normal, or if the abnormalities that were discovered, were corrected, the child was given a five point certificate. The certificates were awarded on a special day, called "child health day."

An unique development and one that showed Dr. Williams' interest in education and public health, is the "Teachers Preventorium" established at the University of Virginia Hospital in 1929-30. Here any teacher in the Public School System can go for a general check up and treatment at a nominal cost.

With the establishment of the Bureau of Vital Statistics in 1912, it soon became evident that maternal and infant deaths were entirely too frequent. A Bureau of Public Health Nursing and Child Welfare was established. Virginia is essentially a rural state and a great number of deliveries at that time took place in inaccessible localities and more than one third of them in the care of midwives. Public health nurses were employed to instruct prospective mothers by visits and by correspondence courses. Attempts were made to instruct the midwives and to regulate their practice and a Committee from the Medical Society of Virginia was asked to appoint a Committee to co-operate with the State Health Department in solving this problem.

Thus the Health Department developed from a $40,000 a year concern to one spending over a million a year. It became recognized as a model and in consequence thereof, Dr. Williams was elected President of the Conference of State and Provisional Boards of Health of North America from 1915-16. Dr. Alan Freeman, his Assistant Commissioner, became professor in the School of Hygiene of Johns Hopkins University, when that school was started, and eventually was dean.

The Virginia Health Department has continued to grow along the lines that Dr. Williams laid down. Last year it expended $6,484,415, and the reduction in the death rate and in the incidence of communicable diseases justifies this expenditure. Just as Sir Christopher Wren needs no monument so long as St. Paul's stands on Ludgate Hill, so Ennion G. Williams will be remembered as long as the Virginia State Health Department continues to grow and to function efficiently. Truly, Dr. Ennion Gifford Williams deserves the title "Statesman of Public Health."

The Rise and Fall of the "Springs" in Virginia

THE high, cool valleys of the Alleghany mountains in Virginia are dotted with mineral springs from Berkeley Springs near the Potomac to the Yellow Sulphur in Montgomery County. In the 18th century each spring was the seat of a resort which consisted essentially of cottages grouped about the spring, a dining room and a dance hall. Some of the more ambitious had swimming pools. Almost completely surrounding this highland area were broad, rich agricultural flat lands spread out like a hugh sycle—pleasant enough places to live in the winter, spring and fall, but uncomfortable and unhealthy in summer. This land was cursed with great epidemics of yellow fever that extended

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up the Mississippi Valley or along the Atlantic seaboard. Occasionally, there were threats of the dread Asiatic cholera. In addition it was plagued with endemics of typhoid fever and malaria. In dog days it was impossible for white people, especially people of quality, to live in this land.

For over a century, "curing" and mating, gambling and dancing went on furiously at the Virginia springs. From the deep South the people came by steamboat to Guyandotte (now Huntington, W. Va.) and overland by stage coach. From the South Atlantic states they came by carriages with house servants and trunks in wagons. From the East they came by canal boat to the end of the James River and Kanawa Canal above Lynchburg. At the height of the season it was as hard to get accommodations in this vast area as it is to find a room in New York City today. Even in the hard days of Reconstruction the traffic kept up although the tempo of the amusements changed somewhat. In several generations after the War social leadership passed from Southern Belles to Northern and Western matrons of great wealth. Entertainments became extravagant—favors from Tiffany and reporters from northern newspapers. Private cars of R.R. presidents stood on special sidings. With the end of the century all this disappeared as suddenly and as mysteriously as the great flocks of wild pigeons that used to darken the skies of Transylvania. Of the few "springs" that remain, none is a "watering" resort in the old sense. One is a sort of Chautauqua. The Homestead and the Greenbrier have been rebuilt and are run like European spas.

Many explanations have been advanced for this phenomenon. Dr. John P. Little, writing in 1851, has this to say about the Virginia resorts. "The travel to and from the distant spring, where bad water is drunk for the sake of good health, strengthens the languid frame, and quickens the feeble pulse; and the valuable qualities of the water as a fountain of health are trumpeted forth by those who have received the benefits of the journey, the rough jolting and the plain fare, with the necessary scramble to obtain a proper share of it. The change of scene, absence of care, new associates, and firm belief in the virtues of the spring work wonders in the curable cases of disease.

"A railroad leading up to one of these health-giving springs, would very much destroy its reputation, by making it easy of access, and thus preventing the beneficial journey." He says the monks understood this principle in the middle ages when they put their hospices in inaccessible places. Percival Reniers, who has written a delightful book about the Virginia Springs, attributes the disappearance of these resorts to the coming of the automobile. However, the springs "folded up" fully a decade before the automobile came into use. Dr. James P. Baker is nearer the truth when he attributes the failure of the springs to the improvement of public health in the country as a whole and especially in the South. It was in the Spanish-American War that the house fly was incriminated as a carrier of typhoid bacilli. He was shown to be a greater menace in country districts than either water or milk. The brilliant researches of the Walter Reed Commission, right after the war, when the role of the mosquito in the spread of yellow fever was proven, is well known. The public learned to swat the fly, screen the home and drain rain puddles and the summer migration to the mountains was no longer necessary. Resorts at the nearby seacoast became possible.

A dissertation might be written about the changes in our ideas about individual hygiene that took place at about this time. The public lost interest in water. Lithia water as a solvent of uric acid is no longer discussed. Mineral water from famous springs or wells is no longer shipped over the country. Gouty diathesis as a topic of

conversation was replaced by a discussion of the kind of climate that is good for sinus trouble. Now the talk is of health foods—overweight and undernourishment. The girls have lost interest in crinoline and organdy and exhibit instead a well distributed coat of tan. Glamour has no place in modern makeup.

Henry Satterwhite Johnson

AS WE are about to go to press the sad news comes of the death of our Executive Secretary-Treasurer and Director of Public Relations. Mr. Johnson died May 23rd in the early morning hours. In the past year and a half he had had several coronary attacks. He went to the hospital several days ago, but failed to respond to treatment as he had done in previous attacks.

His connection with the Medical Society of Virginia began in March 1946 when he was appointed Director of Public Relations. Previously, he was connected with the State Department of Education. At the time of his appointment he was a member of the Virginia Legislature, representing Fluvanna, Goochland, and Louisa Counties. By nature and training he was well qualified. His charming personality and his interest in people made him welcome all over Virginia. It was our privilege to take a great many trips with him in the course of his duties and we have first hand information on this point. He knew and loved Virginia and as we drove along the highways there was scarcely a house or a locality about which he did not have an interesting anecdote. In January 1950, the Secretary-Treasurership of the Society was added to his duties. Mr. Johnson's outstanding work in medical public relations was well known at the national level. As chairman of a Committee for finding locations for physicians in rural communities he outlined plans which have been followed for the past several years and which have gained for Virginia national recognition. At the present time the Committee is in touch with 60 physicians who are seeking locations.

Mr. Johnson married Miss Lucy Bralley, his Secretary, December 22, 1950. They were busily engaged in establishing a home near Hylas in Goochland County. Fortunately for the Medical Society of Virginia Mrs. Johnson continued in her secretarial position after marriage. We extend to Mrs. Johnson our deepest sympathy.

Floral Eponym

Tradescantia

JOHN TRADESCANT, (1567?-1637)

JOHN TRADESCANT (1607-1662)

Tradescantia is a genus of 40 species of American plants, mostly tropical. T. fluminensis is the well known wandering jew. T. virginiana is the common spiderwort.

John Tradescant, father and son, were English gardeners and travelers. They put together the earliest museum of which there is a full record. In 1637 the younger Tradescant was in Virginia getting plants and shells for the collection at South Lambeth. He published a catalogue in 1656 as *Museum Tradescantium: or a collection of rarities preserved at South Lambeth near London.* This collection passed in 1694 to Elias Ashmole who installed it at Oxford. "Remnants of this collection have been permitted to survive there. Among them are couple of fragments of what Tradescant called a 'Dodar from the Island of Mauritius: It is not able to flie being so big'."

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THE PRESIDENT'S MESSAGE

THE Medical Society of Virginia has been quartered in the Richmond Academy of Medicine Building at 1200 East Clay Street, since September 20, 1932.

The offices were comfortable and the environment pleasant, but the functions and activities of the Society simply out-grew the available space.

Within the past few years, three or four people were forced to work in a room with supplies piled high along the walls, on the desks, etc. It simply became necessary to have larger quarters. Efforts were made to find rental space in that vicinity. The Council finally decided that it would probably be best in the end to purchase property to accommodate the headquarters offices. This was done and we moved into 1105 West Franklin Street, April 1.

In this, you have a piece of property and accommodations with which everyone who has seen it seems pleased. It is estimated that the property would bring the purchase price anytime we wish to sell.

The first official Council meeting in the new quarters was held Thursday, April 26. All members are urged when in Richmond to stop by and pay your official home a visit.

One of the first topics of discussion at the recent Council meeting was the question of "kick-backs" or rebates by optical companies to eye specialists, which had been given so much publicity by the Richmond and Norfolk newspapers during the past few weeks.

Dr. Malcolm H. Harris of West Point, Chairman of the Ethics Committee, reported to the council for that Committee April 26, on the committee's consideration of current references to kick-backs.

The Council approved that which had been done by the Ethics Committee and directed a continuation of its work of getting facts regarding kick-backs to be reported by the Committee to Council at its next meeting. If any member of the State Society has any knowledge of the existence of such an arrangement involving a physician, it is his duty to report the case to the Ethics Committee.

While in Richmond your president had the pleasure of attending a part of the first Scientific Assembly of the Virginia Academy of General Practice. They had a very fine and attractive program with excellent attendance. All present seemed to be very enthusiastic over the success of the first session.

This Assembly was patterned after the mid-year meeting of the A.M.A. which meets the first week in December of each year. This meeting was gotten up primarily for the General Practitioner. It was a brilliant success from the start. We wish for them a bright and glorious future.

While attending this assembly, I had the pleasure of talking with Mrs. A. B. Gravatt, Jr., Chairman of "Today's Health" from the Woman's Auxiliary. She stated she was very much discouraged over securing subscriptions. According to her list there are only 74 subscribers in the entire state. There should be at least two thousand. Some of you may recall that several years ago the A.M.A. requested the Woman's Auxiliary throughout the country to sponsor the sale of the old Hygeia, changed two years ago to "Today's Health".

This magazine should be on the reception room table of every physician. It is one medium we have of reaching the general public in language they can understand.

You may not like all the articles; you do not like all the chapters of any book or all the articles in any medical journal, but you like some, and benefit from reading many.

I heartily recommend "Today's Health". It costs only a dollar and a half. It will repay you many fold. Send in your subscription today.

C. L. HARRELL, M.D.

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SOCIETY PROCEEDINGS

The Southwestern Virginia Medical Society

Held its regular Spring meeting on April 20, at the General Shelby Hotel in Bristol, with the president, Dr. J. P. King, presiding. There were forty-five present for the afternoon session and ninety-nine for the banquet. At the afternoon session the following papers were presented: Dr. O. O. Smith, Marion—"Arteriovenous Fistulae and Aneurysms of the Extremities"; Dr. W. R. Chitwood, Wytheville—"Shoulder-Hand Syndrome, Complicating Myocardial Infarction"; Dr. R. Earle Glendy, Roanoke—Moderator of a panel discussion "ACTH and Cortisone", participated in by Drs. Ernest Dixon, Don Greever, J. A. Chitwood, Andrew Giesen, W. S. Schiff, and N. J. Chew.

Following the scientific session, a short business meeting was held at which delegates and alternates to the coming meeting of the State Society were elected. A committee was appointed to the study and make recommendations for revisions of the by-laws for the society.

The Woman's Auxiliary of the Southwestern Virginia Medical Society met at the same time and the ladies attended the banquet.

A social hour preceded the banquet after which Dr. A. J. Oberlander of The National Life Insurance Company of Vermont, spoke on "Insurance Medicine". He was followed by Dr. Gordon McNeer of New York City, who spoke on "The Diagnosis and Treatment of Cancer of the Stomach".

The next meeting of the society will be held in September at Roanoke in conjunction with the Roanoke Academy of Medicine.

Dr. Richard C. Potter of Marion is secretary of the Society.

The Fairfax County Medical Society

Had its regular meeting on April 10th.

The chief topic of discussion was the Medical Plan for Falls Church and Fairfax County in the Civilian Defense Program. The Society approved the following basic operation plan: First Aid Stations are to be established throughout the city and county at strategic population centers; for example, schools and volunteer fire departments. These Stations will be manned by a medical assistant, 2 nurses, 2 nurses aides, 1 record clerk, 8 litter bearers, 4 messengers, and a messenger leader. Physicians or dentists will be dispatched to any of these Stations from a Headquarters Station located in the Falls Church Fire House. In addition Clearing Stations will be established to determine disposition of cases and to give more definitive care than at the Aid Stations. These will be manned by 2 physicians, 2 medical assistants, 15 nurses, 30 nurses aides, 1 record clerk, 4 litter bearers and 4 messengers.

The Society believes that because of the limited number of doctors available and the geographical pecularities of this area this plan will operate best, giving most coverage and allowing for quick movement of personnel to areas needed.

In addition to the above plan a committee has been established by the Society to draw up recommendations in regards to the continuance of normal medical care in the case of an emergency.

CLAUDE E. COOPER, *Secretary*

A Clinical Session,

For the benefit of members of Mid-Tidewater and Northern Neck Medical Societies, under the auspices of the Department of Clinical and Medical Education of the Medical Society of Virginia, was held at the Yacht Club, Tappahannock, Virginia, April 11, 1951.

Classes began at 2:00 P.M. and continued to 9:00 P.M., with social hour and supper from 5:30 to 7:00 P.M.

The speakers for this occasion included Dr. Hudnall Ware, Jr., Dr. Edwin L. Kendig, and Dr. Thomas W. Murrell, Jr., all of whom are on the teaching staff of the Medical College of Virginia.

The combined attendance from both societies numbered thirty six. It was felt by all who attended that such clinical sessions are well worth while, in so far as it affords the physician who practices medicine in the rural area an excellent opportunity to keep abreast with modern methods of diagnosis and treatment of diseases he encounters during his days work.

Alexandria Medical Society.

New officers of this Society, who were installed on May 10th, are: President, Dr. Alfred Abramson; vice-president, Dr. C. A. Hudson; and secretary-treasurer, Dr. F. Preston Titus.

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NEWS

The Virginia Academy of General Practice

Held its first scientific assembly in Richmond on April 26th and 27th. One hundred and ninety physicians from throughout the State were registered. Dr. Ira Hancock, Creeds, is president of the Academy, and Dr. E. E. Haddock, Richmond, was chairman of the committee on local arrangements.

Dr. Raymond B. Pinchbeck, Richmond, was the banquet speaker, his subject being "A Layman Looks at His Doctor". Dr. Philip Thorek, Chicago, Illinois, was the guest lecturer and his subject was "The Acute Abdomen". Others participating in the program were Drs. E. L. Kendig, Jr., Eric C. Schelin, Henry Spalding, Geoffrey T. Mann, Kinloch Nelson, C. E. Rodriguez, Charles M. Caravati, Virgil May, and W. T. Thompson, Jr., all of Richmond; Dr. William H. Kaufman, Roanoke; Drs. H. B. Mulholland, William Parson, and Walter Klingman, of Charlottesville; Drs. Margaret and R. E. Glendy, Roanoke.

It was decided to make this an annual affair and the next assembly will be held in Roanoke, May 6 and 7, 1952, with Dr. John O. Boyd, Jr., as general chairman.

The Virginia Society of Ophthalmology and Otolaryngology

Held its thirty-second annual meeting in Roanoke on May 12th, with the president, Dr. Charles A. Young of Roanoke, presiding. The guest of honor was Dr. Halstead S. Hedges, Emeritus Professor of Ophthalmology of the University of Virginia, who has been associated with the University since 1890. Dr. Fletcher D. Woodward, Charlottesville, gave the lecture in honor of Dr. Hedges. Dr. Louis H. Clerf, Philadelphia, guest, spoke on Paralysis of the Larynx. Dr. J. H. Dunnington of New York, the other guest, was prevented by flying conditions from being present. Case reports were given by Roanoke doctors as follows: Amblyopia Exsanguinata by Dr. William F. Hatcher; Rhinoplasty by Dr. Frederick D. White; and Development of Cataracts Following Electric Shock by Dr. Mortimer H. Williams.

Dr. Frederick Morrison, Lynchburg, who was unable to accept the presidency, was made an honorary life member as also was Dr. Hedges. Dr. R. H. Courtney of Richmond was elected President, Dr. A. D. Morgan of Norfolk, president-elect, Dr. H. B. Stone, Jr., of Roanoke, vice-president, and Dr. G. S. FitzHugh of Charlottesville was named secretary-treasurer. The next meeting will be at Staunton in May 1952.

The Psychological Reaction to the Unaesthetic Nose.

A paper with the above caption, by William Lawrence Gatewood, M.D., of Richmond, appeared in the May issue of the Monthly, but by error failed to appear in the index of that number. We regret this error but it will be listed in the general index.

The Neuropsychiatric Society of Virginia.

The Spring meeting was held on May 2, at the Keswick County Club, Keswick. The following officers were elected for the ensuing year: Dr. R. Coleman Longan, Jr., Richmond, President; Dr. James B. Funkhouser, Richmond, Vice President; Dr. Frank Strickler, Roanoke, Secretary-Treasurer; Dr. Walter O. Klingman, Charlottesville, Executive Officer; and Dr. Granville Jones, Williamsburg, Executive Officer.

The State Board of Medical Examiners of Virginia

Will hold its next meeting at the Richmond Hotel in Richmond, June 13, 1951. The examinations will be held in the same hotel June 14, 15, and 16th. All papers pertaining to the examination or to matters to be brought before the Board must be on file in the office of the secretary of the Board, Dr. K. D. Graves, 631 First Street, S. W., Roanoke, Virginia, not later than June 4, 1951.

News from the Department of Medicine, University of Virginia.

Dr. Lester A. Wilson, Jr., Instructor in Obstetrics and Gynecology, Duke University, has been appointed Assistant Professor of Obstetrics and Gynecology at the University of Virginia, effective May 1.

Dr. Wilson received his B.S. degree from the College of William and Mary, 1938, and his M.D. degree from the Medical College of South Carolina in 1942. After completing his internship at the Gorgas Hospital, Panama Canal Zone, and service

in the Army Medical Corps, he served as resident in obstetrics and gynecology at the Duke University Hospital until appointed to the faculty.

Edwin W. Pullen, Instructor in Biology, University of Virginia, has been appointed Instructor in Anatomy, effective September 1.

Mr. Pullen received his A.B. degree, 1943, from Colgate University, where for two years he held the President's Scholarship. He received his M.S. degree in zoology from the University of Massachusetts in 1948 and since that time has been associated with the University of Virginia as Service Fellow and Instructor in Biology.

Two Virginia physicians have been appointed to top posts in the Red Cross National Blood Program: Major General David Grant, Virginia Beach, and Dr. Russell Haden, Crozet. Dr. Grant was named Director of the American Red Cross National Blood Program and Medical Director of the American Red Cross. He succeeds Dr. G. F. McGinnes, Washington, who has resigned. Dr. Grant will direct all phases of the Red Cross blood program, which provides blood for military and civilian needs and civil defense. Dr. Haden has been named associate director of the ARC National Blood Program. Both Dr. Grant and Dr. Haden are graduates of the University of Virginia Department of Medicine.

Elected to the University of Virginia chapter of Alpha Omega Alpha at its April meeting were Dr. G. Slaughter Fitz-Hugh, Professor of Otolaryngology, University of Virginia, and Rear Admiral H. Lamont Pugh, Surgeon General of the Navy. Initiates from the third-year medical class were Charles Crowder, Jr., South Hill, Va.; Meade Edmunds, Jr., Petersburg, Va.; G. L. Gold, New York City; and Nelson Richards, Charlottesville, Va.

The 50th anniversary of the founding of the University of Virginia Hospital School of Nursing was celebrated in Charlottesville, May 23, 24, 25.

When the School was founded in 1901 the University Hospital was a building of 20 beds. Nurses attending the celebration were invited to tour the present Hospital of 500 beds. Two of the four graduates of the first class attended the banquet, alumnae meetings and graduation ceremonies.

President Colgate Darden awarded diplomas, May 25, to 60 candidates, who were presented by Dr. Vernon Lippard, Dean of the Department of Medicine. Dr. Richard Ackart, Director of the University Hospital, presented the hospital alumnae pin.

King's Daughters' Hospital Opened.

Staunton's new King's Daughters' Hospital was opened on April 15th. The modern hospital at the northern outskirts of the City replaces an outgrown predecessor which had served the community since 1905. It was built at a cost of $2,500,000 and is a 117-bed structure. A feature of the new hospital is an outpatient department which contains the following clinics: eye-ear-nose-throat, dental, physical therapy, genito-urinary, basal metabolism, electrocardiogram and pneumothorax. Also included are fully equipped operating rooms, highly technical laboratory facilities and an x-ray department. There are two major operating rooms, two delivery rooms and two labor rooms, and the nursery is designed to accommodate 28 babies.

J. Shelton Horsley Memorial Lecture.

The fifth annual J. Shelton Horsley Memorial Lecture was given at the Richmond Academy of Medicine on April 24th by Dr. Frank E. Adair of Memorial Hospital, New York City.

The Virginia Radiological Society

Held its annual meeting at McGuire Veterans Hospital in Richmond on April 23rd, with an attendance of 75 doctors. Dr. Aubrey O. Hampton, Washington, D. C., delivered the principal address on "Roentgen Manifestations of Acute Pulmonary Disease"; Dr. Samuel Richman, Richmond, read papers on "Roentgen Findings in Early Carcinoma of the Lungs" and "Single Dose X-Ray Combined with Nitrogen Mustard Therapy in Lymphomas"; and Dr. William J. Barry, Jr., Richmond presented a paper on "X-Ray Findings in Acute Pancreatitis".

Dr. William P. Gilmer, Clifton Forge, is president of the Society, and Dr. P. B. Parsons, Norfolk, is secretary.

The Virginia Obstetrical and Gynecological Society

Held its 1951 Travel Clinic in Chicago, April 9-10. The program was put on by Dr. Frederick Falls of the University of Illinois and Dr. Herbert Schmitz of Loyola University.

Present were the following: Dr. William Bickers, Richmond; Dr. Harvey Bland, Newport News;

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Dr. Milton Bland, Norfolk; Dr. Chester Bradley, Newport News; Dr. William Byrd, Norfolk; Dr. Adrien Carson, Jr., Richmond; Dr. William Cox, Richmond; Dr. Alfred Garnett, Norfolk; Dr. Pembroke Grove, Winchester; Dr. Paul Halter, Arlington; Dr. Gwendolyn Hudson, Richmond; Dr. John Kight, Norfolk; Dr. John Nokes, Charlottesville; Dr. Harrison Picot, Alexandria; Dr. John Saunders, Lynchburg; Dr. William Thornton, Charlottesville; Dr. William Varner, Franklin; Dr. Meyer Vitsky, Richmond; Dr. A. D. F. White, Lynchburg; and Dr. James Whitfield, Richmond.

The following guests were present: Dr. Thomas Bell, Staunton, and Dr. Charles Merritt, Charlottesville.

The American Gynecological Society

Held its Diamond Jubilee meeting on May 7-9, at the Waldorf-Astoria in New York City. Dr. Walter Dannreuther of New York City was elected president and Dr. John I. Brewer of Chicago secretary. The next meeting of the Society will be held on May 12, 13 and 14, 1952 at Hot Springs, Virginia.

The Virginia Society for Pathology and Laboratory Medicine

Held their annual meeting at the Medical College of Virginia, Richmond, on Saturday, March 10. The Society unanimously voted to re-state the position of the members in providing free laboratory service for those patients unable to pay.

Officers elected for 1951 are: President, Dr. William D. Dolan, Arlington; Vice-President, Dr. Arnold Rawson, Norfolk; and, Secretary-Treasurer, Dr. Martin L. Dreyfuss, Clifton Forge.

Dr. S. D. Gardner

Has been appointed Henrico jail physician to replace Dr. A. P. Traynham who resigned after twenty-seven years of service. Dr. Traynham plans to establish a practice in Sweet Springs, W. Va.

Dr. R. Finley Gayle, Jr.,

Richmond, was re-elected secretary of the American Psychiatric Association at its recent annual meeting in Cincinnati.

For Sale.

Ideal location for physician desiring light practice, at Ceres, Bland County, Virginia. A new seven room office-apartment, one floor, well constructed, all modern conveniences. One physician in county at present. Located in beautiful farming valley with excellent hunting and fishing. Sacrifice price for quick sale. Write P. O. Box 17, Spotsylvania, Virginia. (*Adv.*)

Office Space for Rent.

Doctor's or Dentist's office available immediately, corner of Harrison and Franklin Streets, Richmond. Call 5-4387. (*Adv.*)

Locum Tenens Wanted.

July or August, general practice or internal medicine, graduate of Medical College of Virginia, university internship, 21 months Army, 1 year residency in internal medicine. Married, no children, car, highest references. Address "Locum Tenens", care the Virginia Medical Monthly, 1105 West Franklin Street, Richmond 20. (*Adv.*)

OBITUARIES

Dr. Estridge Peterson White,

Widely known physician of York County, with his home at Odd, died April 19th, having been in ill health for two years. He was sixty-seven years of age and a graduate of the Medical College of Virginia in 1913. Dr. White was a former member of the York Board of Supervisors and retired as president of the First National Bank of Poquoson in 1950, having served in that position for twenty-five years. He served for many years as roentgenologist at the Dixie Hospital, Hampton. Dr. White had been a member of the Medical Society of Virginia for twenty-six years. A son, Dr. Stanley Wise White of Poquoson, survives him.

Dr. Herbert Wilson Rogers,

Prominent physician of Norfolk, died May 8th. He was born in Nassawadox in 1892, and received his medical degree from the University of Maryland in 1916. Dr. Rogers had served for many years as a member of the Norfolk School Board and was active in civic affairs. He has been a member of the Medical Society of Virginia for thirty years. His wife, two sisters and four brothers survive him.